"COINCIDENCE OF HEPATITIS B, HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD O.P."

Dissertation submitted in partial fulfilment of the requirements for the degree of

M.D. (DERMATOLOGY, VENEREOLOGY & LEPROSY)

BRANCH XX

MADRAS MEDICAL COLLEGE

CHENNAI-600 003

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY 2018

CERTIFICATE

Certified that this dissertation titled **"COINCIDENCE OF HEPATITIS B, HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD O.P."** is a bonafide work done by **Dr.V.LAKSHMI PRIYA,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2015 – 2018. This work has not previously formed the basis for the award of any degree.

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The dissertation entitled "**COINCIDENCE OF HEPATITIS B, HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD O.P"** is a bonafide work done by **Dr. V.LAKSHMI PRIYA** at Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2014-2017 under the guidance of **Prof. Dr. S. KALAIVANI M.D., D.V,** Professor and Director, Institute of Venereology, Madras Medical College, Chennai -3.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Dermatology, Venereology and Leprosy (BRANCH – XX)

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I, **Dr. V.LAKSHMI PRIYA** solemnly declare that this dissertation titled "**COINCIDENCE OF HEPATITIS B, HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD O.P."** is a bonafide work done by me at Madras Medical College during 2014-2017 under the guidance and supervision of **Prof. S.KALAIVANI, M.D., D.V.,** Professor and Director, Institute of Venereology, Madras Medical College, Chennai-600 003.

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SPECIAL ACKNOWLEDGEMENT

My sincere thanks to **Dr. R.NARAYANA BABU M.D., D.C.H.,** Dean, Madras Medical College, Chennai-3 for allowing me to do this dissertation and utilize the Institutional facilities.

ACKNOWLEDGEMENT

I am grateful to the to P**rof. Dr. S. KALAIVANI, M.D., D.V.,** Director and Professor, Institute of Venereology**.,**for her advice, guidance and encouragement for my study. She has been a source of constant motivation and encouragement throughout the study. I am extremely grateful to her for guiding me throughout the study.

I would like to express my sincere and heartfelt gratitude, Professor and Head of the Department of Dermatology, **Prof. Dr. U.R. DHANALAKSHMI, M.D., D.D., D.N.B** for her kindness and support throughout the study.

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I wish to thank **Dr. S.VIJAYA BASKAR, M.D., D.C.H .,** Additional professor, Institute of Venereology for his guidance.

I sincerely thank **Prof**. **Dr. S. NIRMALA M.D., Professor and Head of Department, Department of Occupational and Contact Dermatitis** for her valuable support.

I sincerely thank **Prof**. **Dr. R. PRIYAVATHANI ANNIE MALATHY, M.D., D.D., D.N.B., M.N.A.M.S.,** Professor of dermatology for her help and support.

I thank **Prof. Dr. S. KUMARAVEL, M.D., D.D.,** Professor of Dermatology for his advice and encouragement.

I thank **Prof. Dr. V. SAMPATH M.D., D.D.,** Professor of Dermatology for his advice and encouragement.

I thank **Prof. Dr. A.RAMESH M.D., D.D., D.N.B.,** Professor of Dermatology for his advice and encouragement.

I am grateful to **Prof. Dr. J. MANJULA, M.D., D.N.B.,** Professor, Department of Dermatology for her invaluable guidance, help and encouragement.

I humbly thank my Co-Guide **Dr.H.DHANASELVI M.D.D.V.L** Assistant professor, Institute of Venereology for his valuable guidance throughout my work. I would like to express my sincere and heartfelt gratitude for the time which they devoted for my research project.

I also thank my Assistant Professors **Dr. P. PRABAHAR, M.D.D.V.L., Dr. C. VIDHYA, M.D.DVL., Dr.R.HEMAMALINI, M.D.D.V.L., Dr.E.BALASUBRAMANIAN,M.D.D.V.L., Dr.K.GAYATHRI, M.D.D.V.L., and Dr.R.SNEKAVALLI M.D.D.V.L.,Dr S.TAMILSELVI M.D.D.V.L, Dr T. VANATHI, M.D.D.V.L,** Institute of Venereology for their able guidance

I thank **Dr. DILLIRANI, M.D.,** Professor of Serology and **Dr. HEMALATHA, M.D.,** for her help and support

I extend my gratitude to **Dr.R.MADHU, M.D., (DERM)., D.C.H., Dr.SAMUEL JEYARAJ DANIEL,M.D.D.V.L. Dr.V.N.S.AHAMED SHARIFF, M.D.D.V.L ,Dr.B.VIJAYALAKHSMI, M.D.D.V.L., Dr.K.UMAMAHESWARI, M.D.D.V.L., Dr.R.MANIPRIYA, M.D.D.V.L., D.C.H., and Dr.C.L.CHITRA, M.D.D.V.L Dr. K.DEEPA, M.D.D.V.L,** Assistant professors, Department of Dermatology for their kind support and encouragement.

I am thankful to my colleagues for their support throughout the study. I am also grateful to all paramedical staffs for rendering timely help to complete my study. Last but not the least I am profoundly grateful to all patients for their co-operation and participation in this study. They have been the principal source of knowledge which I have gained during the course of my clinical research.

CONTENTS

Introduction

INTRODUCTION

Genital ulcer is defined as the breach in the genital mucosa and or the skin. Genital ulcer disease constitutes about 1% to 70% of the sexually transmitted diseases in various parts of the world[1]. The incidence of genital ulcer disease varies from region to region, influenced by the variation in the prevalence of causative agents, sociocultural factors, the sexual behavior of the population, economy and civilization.

In the whites and in developed countries, herpetic ulcers are common. In Blacks and Hispanics, syphilis and chancroid are common. In developing countries chancroid is common .In both developing and industrialized countries of the world, syphilis is common[2]. In some parts of Asia, most importantly South India, Donovanosis is seen.[3] Various studies in India show a rise in the incidence of herpes, and in some studies, the prevalence of herpes has exceeded the prevalence of syphilis and chancroid.[4-6]

Genital ulcers are frequently reported in men, as they are easily detected in males compared to females. Males act as the reservoir of infection constantly infecting the male and the female population. Genital ulcers which are symptomatic have an equal incidence in males and females, whereas those which are asymptomatic are under reported in females.

Females with asymptomatic genital ulcers are the reservoir of infection and they form a significant risk group infecting male partners.[2] Due to the increasing coinfection with HIV and mixed infections, the morphology of the genital ulcers are altered and the prototypical description of the ulcers may not be present.[7] A secondarily infected syphilitic chancre can mimic chancroid, and herpes and chancroid coinfection may cause diagnostic difficulties.

The ulcers be confined to genitalia or may be found in the extragenital sites because of changes in the sexual behavioural pattern. Due to the self medication or the contamination of the ulcers, the demonstration of the etiological agents causing the ulcer becomes difficult. In case of early lesions, the bedside and laboratory investigations are fairly sensitive and specific.[2]

Review of Literature

REVIEW OF LITERATURE

Etiological classification of genital ulcer disease[8]

Genital ulcers can be due to venereal diseases more commonly and rarely due to other dermatological or systemic conditions.

The venereal causes of genital ulcer are:

- Syphilis
- Chancroid
- Herpes simplex
- Granuloma inguinale
- Lymphogranuloma venereum.

The non venereal causes of genital ulcers are:

- Traumatic ulcers
- Vesiculobullous skin diseases
- Adverse Drug Reactions like Steven Johnson Syndrome and Toxic Epidermal Necrolysis
- Behcets disease
- Erosive Lichen Planus
- Reiters disease
- Erythema multiforme
- Infections due to pyogenic organisms, yeast and non specific spirochaetes,

- Crohn’s disease
- Premalignant conditions like Pagets disease, Bowens disease, Erythroplasia of Queyrat
- Neoplasms like Squamous Cell Carcinoma and Basal Cell Carcinoma
- Non specific causes.

HIV

The Human Immunodeficiency Virus is a single stranded RNA virus and belongs to retroviridae family. HIV seems to have been transmitted to humans from two non human primates infected with simian immunodeficiency virus. HIV-1 is the isolate transmitted from chimpanzees, and those from sooty mangabey monkeys are known as HIV-2. HIV-1 is the major cause of the disease worldwide.

The clinical spectra is similar in both infections, except for the fact that HIV-2 has a lower infectivity, with a longer incubation period and slower progression. Most of the infections have been acquired through sexual contact, 80.8%, and about 5.1% associated with injecting drug use and 5.5% through transfusion of blood and blood products and less than 1% was attributed to mother to child transmission of the disease. In men having sex with men, HIV infection, is commonly contracted through unprotected anal intercourse.

Within a period of 2 to 4 weeks, seroconversion illness develops and lasts for about 2 to 3 weeks. It is followed by early asymptomatic HIV disease, which lasts for about 8 years on an average, followed by the development of late symptomatic HIV disease. This period indicates the advanced immunosupression and increase in the incidence of opportunistic infections. Finally the patient develops advanced disease. The infection is predominantly sexually transmitted and hence, genital mucosa is the major portal of entry for HIV virus.

Mucosal surfaces are rich in langerhans cells-the dendritic cells that can trap viral particles. Within hours after exposure of genital mucosa to the virus, the replicating virus is found in the regional lymph nodes. The primary viremia follows, leading to dissemination of the virus which leads to lymphoid tissue seeding all over the body causing acute HIV infection.

During the primary viremia, very high titres of plasma viral load can be detected. Later, there is a development of virus specific immunity, and the virus becomes undetectable. The memory CD4+ lymphocytes are preferentially infected by HIV and they serve as the coreceptors for the virus. Most of the infected CD4 cells die within a few days of infection.

The disease progression depends on host factors. In case of older people, the progression of the disease is more rapid. Seropositive intravenous drug users are at increased risk for the rapid progression of the disease. Coinfection with

hepatitis C causes a rapid progression of the disease. Severe malnutrition found in developing countries is associated with a rapid progression of the disease. The infection is divided into four stages, the Acute seroconversion illness, Asymptomatic stage, Early symptomatic stage, the Late symptomatic and the advanced stage of HIV infection (AIDS). “Acute HIV infection” or “Primary HIV infection” is the first stage of HIV disease.

Majority of the transmission of infection occurs across the mucosal surface such as anorectal, vaginal and less commonly, the oral mucosa. The receptive cells for the HIV are found in the lamina propria of cervico vaginal, rectal, foreskin, urethral and oral mucosa in studies conducted in primate models. There is increased expression of CCR5 chemokine coreceptor in the mucosal cells. During the active infection, profound depletion of CD4+ T cells occur, which is followed by infection of other cell types including the dendritic cells, macrophages and resting T cells.

Resting T cells form the major reservoir of latent HIV infection. Following local infection, the dendritic cells play an important role in the dissemination of infection. The HIV antigens infecting the peripheral tissue is brought into contact with T lymphocytes in the draining lymph nodes through the dendritic cells, and hence plays an important role in the generation of immune response against the antigens.

HIV enters the immature dendritic cells at the mucosal and epithelial surfaces and limited replication occurs in these cells, till they are carried to T cells in the lymph nodes. HIV bound to the unique lectin on dendritic cells known as DC-SIGN, remains viable for many days which provides an additional mechanism for the regional lymph nodal spread. This stage of infection, where there is a rapid rise in plasma viral load and increased number of viral particles in genital secretions, is significant from a public health point of view because of the high infectivity.

Any breach in the genital mucosa or the presence of increased inflammation due to ulcerative genital disease, or urethritis or cervicitis is associated with an enhanced sexual transmission of the infection. After the establishment of HIV infection in the lymphoid tissues, the viral loads fall in the following weeks, due to the cellular immune response mounted against the virus.

Within days to weeks following exposure, the symptoms and signs of primary HIV infection may appear, lasting from a few days to 10 weeks, with less than 14days in a majority of individuals. Fatigue, fever and rash are the three most common symptoms, but nausea, vomiting, diarrhoea, night sweats, pharyngitis, lymphadenopathy, headache and myalgia can also occur. Diffuse maculopapular involving the trunk, face and the limbs with palms and soles involvement helps in the diagnosis. The primary HIV infection may rarely present with genital ulceration.

Diagnosing primary HIV infection is significant in controlling the propogation of the epidemic. The detection of p24 antigen in the plasma or the serum is employed for the detection of infection during the seroconversion period. Identification and counseling of patients with primary HIV infection may have an important role in preventing the spread of infection. As the spectrum changes from acute syndrome to advanced disease, the CD4+ count declines, correlating with degree of immunosupression. When the CD4+ count is greater than 500 cells/ µl, opportunistic infections causing systemic symptoms are less likely.

Between 200 and 500 cells/µl, sinusitis and bacterial pneumonia, tuberculosis are common. When the count declines to less than 200 cells/µl, many opportunistic infections like Candidiasis, Strongyloides stercoralis, Cryptosporidium parvum, Pneumocystis jiroveci pneumonia, disseminated Mycobacterium avium complex, Cryptococcosis, Toxoplasmosis and Cytomegalovirus retinitis occur. The life threatening severe infections occur when CD4 cell counts fall below 200/µl. Immune Reconstitution Inflammatory Syndrome is seen in many opportunistic infections, after initiation of Anti Retroviral Therapy.

It is the paradoxical worsening of the clinical status of the patient, after initiation of ART, inspite of improved immune function. NSAIDS, corticosteroids, treatment of the underlying infections and continuing ART, unless the response is life threatening, are the treatment principles in management of IRIS.

More than 90 % of persons infected with HIV have cutaneous signs and symptoms, skin being the most common organ affected in patients with HIV. Dermatalogic diseases in HIV may range from inflammatory, infective or oncogenic disorders. They are very significant, and in most of the situations, they may be the diagnostic markers of HIV infection and also the opportunistic infections.

The skin lesions are atypical and responds poorly to conventional treatment. Most of the cutaneous manifestations, respond spontaneously to ART. The once rare disorders such as Bacillary Angiomatosis, Kaposi Sarcoma and eosinophilic folliculitis are brought to attention because of the HIV epidemic .Lungs are the major targets of HIV infection and pulmonary macrophages play a significant role in pathogenesis of AIDS, being the discrete target cells for HIV.

The patients are at increased risk for opportunistic infections of the lung and neoplasms. Mycobacterium tuberculosis is the most common coinfection in HIV infected patients in South East Asia, especially India. Tuberculosis can occur at any stage, irrespective of CD4 count, though it is common when the count falls below 300 cells/µl.

When the count falls below 200 cells/µl, there is an increased chance of dissemination, with extra pulmonary involvement of central Nervous System, Liver, Spleen and Bone. Primary progressive lung disease is also common. Guidelines should be followed before the initiation of ART since, IRIS is a

significant issue while treating HIV/TB coinfection. Extensive Kaposi sarcoma of the lung in HIV infected patients, reduces the median survival of the patients. Highly Active Anti Retroviral Therapy, helps in the resolution of the lesions. The opportunistic infections of the CNS is more common in patients not on antiretroviral therapy.

Apart from infections, other manifestations like peripheral neuropathy, neoplasms, dementia can also occur in the HIV infected patients. Diarrhoea is the most common manifestation of HIV infection, the opportunistic infections being the most common cause. The clinical presentation differ according to the site of involvement of Gastrointestinal tract. GIT is the common site of HIV associated Non Hodgkins Lymphoma. HIV associated lipodystrophy which includes hyperglycemia, insulin resistance and hyperlipidemia increases the risk of cardiovascular disease.

The incidence of NHL is 60 to 200 times high in the HIV infected people when compared to the uninfected. HAART has led to the decrease in the incidence of Kaposi sarcoma and AIDS related lymphoma. Rheumatic manifestations ranging from non specific inflammation, non inflammatory pain syndromes and autoimmune syndromes like vasculitis, sjogrens syndrome, are more common in the late stage of infection. These disorders are significant in the view of treatment , since the immunosuppressive agents used in the treatment of rheumatological conditions, may rapidly cause full blown AIDS.

Ocular diseases in HIV can practically involve any structure of the eye. In case of involvement of posterior segment, early diagnosis and treatment is essential, since it can lead to irreversible blindness. After the introduction of HAART, the incidence of ocular complications has decreased.[9]

HIV infection and genital ulcer:

The epidemic of HIV in the past few years, has caused a significant impact on the transmission, course and treatment of genital ulcer disease, and GUD also has an impact on the transmission and the course of HIV infection.[2] Regardless of the cause of the ulcer, the mere presence of ulcers are associated with an increase in the risk of transmitting or acquiring HIV.[10] HIV and other STDs have a reciprocal relationship which is called as epidemiological synergy, each altering the transmission and manifestations of other STDs.[11] Genital ulcers could facilitate the transmission of HIV1 by increasing the infectiousness of the index case or the susceptibility of the partner or both.[12,13]

Through different biological mechanisms, the genital ulcers promote the transmission of HIV by increasing the infectiousness and HIVsusceptibility.[14] Increased number of HIV infected white blood cells in genital ulcers contribute to the infectivity cofactor effect.[15] A number of studies from different parts of India have shown an increasing prevalence of HIV in patients who attend STD clinics with genital ulcer disease than in the non ulcerative STD patients.[16-19]

Genital Ulcer Disease has a great impact on the spread as well as the course of HIV. Ulcers cause impairment of the normal epithelial barrier, increased HIV shedding from the genital tract, recruiting and activating the cells susceptible to HIV at the lesional site, thereby promoting the replication of HIV.[20]

HIV also influences the course of other sexually transmitted diseases in reactivating latent infections, modifying the original course of the disease, altering clinical picture, challenges in treatment with failure in conventionally followed treatment regimes, altering serological test results and histopathololological picture, thereby posing diagnostic difficulties.[2] In a case study of four cases of genital ulcers in Nigeria, in HIV coinfection the ulcers were long standing, aggressive in nature, recurrent and associated with systemic symptoms.[21]

Herpes genitalis:

Herpes genitalis is caused by Herpes virus hominis, a DNA virus. It is the most common sexually transmitted pathogen causing genital ulcer worldwide and is the most frequent opportunistic infection in HIV infected adults. The virus is of two types-type1 and type2.[22] The genital lesions are predominantly caused by type 2 virus[23] though type-1 may be isolated from one third of the cases. Herpes genitalis is a persistent infection in the sensory ganglion, running a chronic course, with varying degrees of epithelial expression characterized by asymptomatic or symptomatic recurrences. After a primary infection, virus persists in the sensory nerves which is termed as the latency, during which the host immune system fails to recognize the virus.

The virus is reactivated following various triggers, and travel to the skin or the mucous membrane, and the replication causes the recurrent disease. The shedding of virus occurs even in the absence of lesions clinically-asymptomatic shedding, though the number of viral particles that are shed are very less compared to those from an active lesion.[22] Asymptomatic shedding of virus is responsible for about 70% of transmission.[24] Infection spreads through direct contact with the infected secretions.

The average incubation period is about 5 to 14 days. The first episode of the infection is more severe in nature and associated with systemic symptoms like headache, fever, malaise and myalgia. Painful ulcers are common and lasts for upto 2 weeks. Grouped vesicles are the initial lesions which rapidly become pustular and then ulcerate. Ulcers present as large coalescing areas with polycyclic margins. Localized lymphadenopathy and urethral discharge are common.

New lesions occur in the first 10 days and it takes about three weeks for the complete reepithelization to occur. Shedding of virus usually occurs in the first two weeks. Scarring is not common.[22] Recurrent episodes of genital herpes are milder in nature with a shorter duration of about 5-7 days. The viral shedding period is between 3 to 4 days and complete healing occurs by 7 to 10 days.

Recurrences are more common and earlier in HSV-2 infections. The clinical presentation in recurrences is not typical with grouped vesicles as in first

episode. The transmission of HIV is facilitated by the persistent genital ulcers of HSV.[22] The presence of HSV infection increases the transmission and replication of HIV. Herpes simplex virus infections are more frequent and more severe in HIV patients with immunosupression.[25-27] A synergistic relationship exists between HSV and HIV thereby leading to increased replication of the viruses and increased potentiation of transmission of HIV.[28]

Antiretroviral therapy decreases the severity and frequency of symptomatic genital herpes, but subclinical shedding still continues to occur.[29] Asymptomatic HSV shedding and prolonged and continuous viral shedding is more common in HIV seropositive than seronegative individuals. Perianal HSV shedding is also more common in HIV seropositive individuals. Coexistent HIV infection leads to more frequent and prolonged episodes of genital herpes with decreased response to acyclovir.

Atypical presentations of genital herpes like extremely painful ulcers, hyperkeratotic verrucous lesions, deep progressive ulceration, hemorrhagic and ecthyma like lesions, pseudotumor of tongue, pneumonitis, hepatitis, oesophagitis and disseminated infections which are life threatening are common in coinfection with HIV.[22,23] In HIV positive patients, recurrences are more persistent, as against self limiting lesions in HIV uninfected patients.[22] The diagnosis in typical cases is usually made clinically.

The laboratory investigations available for the diagnosis of herpes simplex virus infection are Tzanck smear, histopathology, culture, serology, and polymerase chain reaction. The Tzanck smear is stained with giemsa and multinucleate giant cells are seen. The test is negative in later stages and has a low sensitivity. The test does not differentiate between HSV-1 and HSV-2. Histopathology is rarely performed. Culture is the definitive means of diagnosis of herpes simplex virus and usually positive in the primary infection and negative in about 50% of recurrences.

Negative results are more common in dry erosions or crusted lesions.[22] Enzyme Linked Immunosorbent Assay, Western Blot, Complement Fixation Test are employed for detection. The current generation of tests have a high sensitivity and specificity.[30,31] Various studies have shown Polymerase Chain Reaction, to be superior to culture in detection of virus from mucocutaneous swabs.[32-34] Antiviral drugs used in the treatment of HSV infections are Acyclovir, Valacyclovir and Famciclovir. In cases of acyclovir resistance, foscarnet, trifluridine and cidofovir are used. Vaccines are used for the prevention of acquisition of infection.[22]

Syphilis :

Syphilis is a chronic sexually transmitted bacterial disease caused by thespirochaete, Treponema pallidum, subspecies pallidum, a thin delicate, motile, tightly coiled organism with tapering ends, belonging to the order, Spirochetales and the family, Spirochetaceae. Humans are the only natural host and the reservoir of infection. The disease is systemic from outset, can involve any structure of the

body, can be completely asymptomatic or can cause florid manifestations, and is a great imitator of many other diseases.

The disease has early or the infectious stage which includes primary, secondary and early latent stages and the late or non infectious stage which includes late latent and tertiary stages. The disease spreads through sexual contact from an infected person who is in the early stage of the disease or transmitted from mother to fetus during primary, secondary or early latent stage. The adverse outcomes of transmission to fetus like still birth and congenital syphilis and the transmission of HIV through the ulcerative and inflammatory lesions of syphilis in primary and secondary stages is of significant public health importance.[35]

Treponema pallidum penetrates through minor breaks in the skin or the mucosa. Studies suggest that treponema pallidum stimulates the fibroblasts to synthesis increased levels of interstitial collagenase, which might help the organism to penetrate the cell.[36] The organism slowly multiplies at the site of inoculation and when the number of organisms reach a threshold level, the primary lesion develops. The ulcer (chancre) is the primary syphilitic lesion and usually appears within 9 to 90 days ,at the site of inoculation, usually on the genitalia. The lesion usually starts as a dusky red and a painless macule of about 0.5-1 cm in size which becomes a papule and the size increases and ulcerates.

The ulcer is painless and non tender, with edges being well defined and regular, the floor being clean with dull and red granulation tissue with the characteristic induration at the base and highly contagious serous exudate present on manipulation of the ulcer. Chancre is single in a majority of the patients..Regional lymph nodes enlargement is seen within 7-10 days, initially unilateral, later becoming bilateral.[37]

The primary chancre heals in an average period of about twelve days. In most patients, the primary chancre heals before the lesions of secondary syphilis. The lesions of secondary syphilis start appearing in approximately 8 weeks from the time of contact, with a range from 1 to 12 months.[38] After the resolution of the secondary stage, the person remains asymptomatic and the stage is termed as the latent stage, during which, positive serology is the only evidence of infection. The latent stage is further divided into early latent, which is infectious and the non infectious late latent stage.[39,40]

The latent stage is of a long period, which might last for many years, or may result in spontaneous cure. The tertiary stage develops eventually, manifesting as cardiovascular or the neurosyphilis or the benign gummatous lesions of the skin and other organs. In this stage of the disease, the treponemes are sparse and the immunological mechanisms play an important role in the pathogenesis.

The ulcerative lesions of primary and secondary syphilis facilitates the transmission of HIV.[35] Primary chancre causes a breach in the epithelium, providing easy access for HIV to the CD4 lymphocytes, which contribute the major proportion of cells infiltrating the lesional site. In HIV and Syphilis coinfection, CD4 counts fall rapidly and the viral load increases rapidly.[41,42]

In a community based study, a 10 times higher incidence of syphilis in HIV seropositive Men having Sex with Men(MSM) compared to HIV negative MSM was found, and this was attributed to behavioral factors and unprotected anal intercourse.[43] In patients with advanced HIV, syphilis presents with unusual features clinically. Persistant chancre, painful chancre, multiple primary chancres, giant primary chancre are common in HIV positive individuals.[44-47]

The persistence of primary chancre and the rapid progression to secondary syphilis can occur and it also increases the transmission of HIV.[48] Malignant secondary syphilis known as Lues maligna, an explosiveand widespread secondary syphilis, starting with a prodrome of fever, myalgia and headache, succeeded by a papulopustular eruption, that rapidly breaks down to necrotic ulcers with sharp margins with hemorrhagic crusts, is found to be 60 times more common in HIV infected people when compared to the normal population.[49] More rapid progression to neurosyphilis can occur in coinfection[50] and asymptomatic neurosyphilis is more common.[51]

The clinical and serological response to treatment of syphilis with penicillin is unaltered in HIV and syphilis coinfected patients.[52] The exudates from the mucocutaneous lesions of early congenital and early acquired syphilis is subjected to dark field microscopy, by which the diagnosis of primary syphilis can be made weeks before the serological tests become reactive. Serology plays a significant role in the diagnosis of syphilis, since treponema pallidum cannot be sustained in cultures. Multiplex PCR assays, which can simultaneously detect treponema pallidum, haemophilus ducreyi and herpes simplex virus 1 and 2 in genital ulcer disease have been developed.[53]

Chancroid:

Chancroid is relatively a common cause of genital ulcer in developing countries like India and Africa.[54] Soft sore, soft chancre and ulcus molle are the other names denoting chancroid. The disease was first described as a separate entity by Ricorde. Chancroid is an endemic disease in many developing countries like Kenya , Zimbabwe and Zambia.[55]

Chancroid is caused by a gram negative facultative anaerobic bacillus called Haemophilus ducreyi. H.ducreyi is strictly a human pathogen, which can naturally infect the skin, mucosa and regional lymph nodes with increased preference for the mucosal epithelium.[56] Low hygienic standards and uncircumcised status are associated with the increased prevalence of the disease.[57]

The disease is more common in males when compared to females and the ratio of male to female incidence ranges from 3:1 to 53:1.[58] The variation in incidence, being higher in males compared to females is attributed to facts like asymptomatic ulcers of cervix or vagina in females with spontaneous healing, lymphadenitis and bubo formation being less frequent in females, and better visibility of the lesions on male genitalia.[56]

Commercial sex workers and people belonging to lower socioeconomic status are at increased risk of acquiring the disease. The disease is mainly transmitted through heterosexual contact, usually the infection being acquired from sex workers.[59] The estimated risk of transmission of chancroid from an infected male to a female who is free of disease during a single sexual act is about 0.35 as against 0.30 in case of transmission from an infected female to an uninfected male during a single sexual act. The total duration of infectivity was estimated to be about 45 days.[60] The minor trauma or the abrasion that occurs during the sexual intercourse is the route through which the organism is inoculated into the tissues.[61] The average incubation period ranges from 1 to 14 days.[62]

The lesion starts as a small inflamed papule with a surrounding erythema on the genitalia, rapidly progressing to a pustule, quickly breaking down to form multiple painful sharply circumscribed foul smelling ulcers with ragged undermined edges. A yellowish, necrotic and purulent exudate covers the floor of the ulcer. On removal of the exudate, a richly vascular granulation tissue is found,

that on scraping or on gentle manipulation, bleeds. Multiple ulcers are seen in about 50% of the cases due to autoinoculation.[56]

The inner surface of the prepuce, frenulum and the coronal sulcus are the common sites infected by the bacilli. Within a span of 1 to 2 weeks, painful inguinal lymphadenitis develops in about 30 to 60 % of the patients. The nodes are unilateral in most patients, become enlarged and matted and suppurates forming an abscess called a bubo, when left untreated ruptures to form a sinus.

The opening of the sinus, breaks down to form a chancroidal ulcer, which can sometimes enlarge to form a giant ulcer.[63] In the presence of chancroidal ulcer, the transmission of HIV is increased.[64,65,66] More frequent HIV seroconversion is seen in heterosexual men suffering from genital ulcer disease than in men without genital ulcer. Haemophilus ducreyi infection evokes a cell mediated immune response and attracts the HIV susceptible cells to the site of ulcer.

The macrophages present at the site of chancroid, have an increased expression of CCR5 and CXCR4, the two important chemokine receptors which are essential for the entry of HIV. The disrupted epithelial barrier along with the up regulated receptors provide a conducive environment for the acquisition of HIV infection.[67] Ulcers bleed during intercourse potentially increasing the viral shedding and HIV infectiousness.[68]

In case of coinfection with HIV, the number of ulcers are more in number and take a longer time to heal. Extensive necrotizing ulcers with multilocular buboes are common in HIV coinfection. Atypical ulcers of larger size and number with extragenital location are also common.[68,69] Decreased responsiveness to the standard single dose treatment regimens are common in chancroid and HIV coinfection.[70,71,72]

Lymphogranuloma venereum:

Lymphogranuloma venereum is a sexually transmitted disease caused by Chlamydia trachomatis biovars L -1,2,3. Sexual transmission is the most common mode of transmission of the disease, but non venereal transmission from ruptured buboes to health care personnel and infection through infected birth canal can occur.[73] LGV is more common in men than women, the asymptomatic nature of the lesions in females being a reason for the under diagnosed state in them.[74]

Commercial sex workers have a significant role in the transmission of the disease. The maximal incidence is in the second and third decade of life, which corresponds to the peak age of sexual activity. The disease is more common among men having sex with men, people belonging to lower socioeconomic class, and sexually promiscuous individuals. LGV is a disease of long course and of destructive nature.

The organism is lymphotropic and initiates the disease process by thrombolymphangitis and perilymphangitis. Inoculation occurs through the abrasion in the skin or the mucous membrane and then the organism gets concentrated in the draining lymph nodes, producing lymphangitis and lymphadenitis.

The proliferation of endothelial cells occur in the lymphatics and the lymph channels in the involved lymph nodes. Enlargement and necrosis of the regional lymph nodes occur. Neutrophils are attracted to the site and it leads to the formation of the quadrangular or the triangular stellate abscess, that is surrounded by macrophages, epitheloid cells and the giant cells. The abscesses coalesce to form a multilocular abscess, which undergoes spontaneous rupture, forming fistulae and sinus tracts.

The inflammation subsides and followed by fibrosis which leads to obstruction of lymphatic channels in the submucous and subcutaneous tissue causing lymphoedema, brawny induration and elephentiasis. The overlying skin and the mucous membrane undergoes ischemic necrosis and ulceration due to compromise in the regional blood supply that occurs secondary to fibrosis. The systemic infection can occur due to hematogenous spread of the organism. The tissue damage that occurs in LGV is mainly due to cell mediated hypersensitivity response mounted against chlamydial antigens.[75]

Clinically the presentation of the disease varies according to the patient's sex, mode of sexual contact, and the clinical stage of the disease. The clinical features are grouped as a spectrum consisting of primary stage, secondary stage or the inguinal syndrome and the tertiary stage or the genito-anorectal syndrome. Apart from these there can be urethro-genito-perineal syndrome and significant ocular and cutaneous manifestations.[76,77]

The incubation period averages from 7 to 12 days for primary stage but can be as long as 6 months. The primary lesion usually occurs over the coronal sulcus, starts as an asymptomatic papule, becomes a pustule and transforms into a herpetiform ulcer.[74]

Bloody discharge, diarrhoea and cramps occur in MSM due to primary rectal inoculation.[78] Secondary stage starts within 2 to 6 weeks after primary lesion, but may be prolonged as long as 4 to 6 months. This stage is characterized by severe proctitis, bubo formation and inguinal multilocular abscess. In MSM, LGV proctitis is common, characterized by perianal ulcers, anal cramps, tenesmus and bloody discharge.

The tertiary stage develops in about 25 % of the untreated patients, more commonly in women. The persistent infection elicits a chronic inflammatory response, and causes proctocolitis, "lymphorroids", which is the hyperplasia of the intestinal and perirectal lymphatics, strictures, rectal stenosis and fistulae.[79]

Ocular manifestations can occur at any stage of the disease due to autoinoculation. The disease can present as iritis, episcleritis, iridocycitis, conjunctivitis and corneal ulcers. Cutaneous manifestations are due to hypersensitivity reaction to LGV antigens, and can present as diffusely spread papules, pustules, nodular lesions, urticaria, erythema nodosum, erythema annulare centrifugum or erythem multiforme.[80]

Lymphogranuloma venereum, being an ulcerative disease, there is an increased transmission of HIV as well as hepatitis C. Most of them were associated with the high risk sexual practices causing anal trauma.[81] In a retrospective analysis of 27 LGV patients in a hospital in Paris, in 6 patients, a concomitant HIV infection was found.[82]

The LGV epidemic among men having sex with men, around 80% were coinfected with HIV[76,77,83] and hepatitis C coinfection was found in upto 18%.[76,84] LGV can be diagnosed by commercially available Nucleic Acid Amplification Test and Polymerase Chain Reaction. Cell culture using cell lines such as baby hamster kidney cells, HeLa 229, McCoy cells are used. Cytology by staining with special stains such as Giemsa, Warthin-starry, Grocott methenamine silver, phosphotungstic acid hematoxylin are also used for diagnosis.[53]

Hepatitis C [81,85,86,87]

Hepatitis C virus is an enveloped virus that belongs to the Flaviviridae family. The HCV genome is a single stranded positive sense RNA, of about 9.4 kb length. Based on the nucleotide sequence diverge, 6 genotypes, HCV 1 to HCV 6 are observed and subtypes are denoted as a,b, etc., This genetic heterogeneity occurs because the viral RNA polymerase which helps in the replication, lacks a proof reading exonuclease, and hence error prone.

The virus is capable of causing a persistent infection in a majority of the patients and hence considered notorious. Such patients are more likely to develop cirrhosis and hepatocellular cancer. The prevalence of chronic hepatitis C worldwide is 0.5 to 2 %, which accounts for 150 million carriers in the world. The distribution of genotypes are different in different parts of the world.

The genotypes HCV 1 and HCV 2 are common in western Europe, West Africa, Australia, Japan and North America. In India HCV 3 is common. HCV 4 is common in Egypt and Central Africa and HCV 5 being common in South Africa and HCV 6 in South East Asia. Prevalence is more common in men, with a progressively increasing incidence with increasing age.

Thalassemia patients, patients with haemophilia, recipients of haemodialysis, injecting drug users, persons who have got tattoos, prisoners, alcoholics have increased prevalence of anti-HCV. In Europe and Japan, the prevalence of chronic HCV infection is very high in patients with chronic hepatitis, cirrhosis and hepatocellular carcinoma. HCV transmission occurs more commonly through blood and blood products.

In the past, HCV accounted for 85% of post transfusion hepatitis that has now decreased to 4 %, due to exclusion of paid donors and the mandatory screening of blood for HCV. HCV also occurs through other routes such as injecting drugs usage, where the sharing of needles and syringes is identified as the risk factor in the transmission of the disease. About 50% to 80% of people involved in injecting drug use, turn HCV positive in a period of twelve months of starting drug use and by 8 years, all are anti HCV positive.

The healthcare workers are at risk of seroprevalence, with the greatest risk among dental surgeons. The health care workers who get exposed to HCV positive blood by accidental needle stick injuries have a serocoversion rate of about 1.8% on an average. In the past, HCV infection was more among patients with renal failure, because of shared hemodialysis machines. Nowadays, patients are routinely screened for HCV infection, and if found positive, hemodialysis is carried out in separate machines designated for infected patients.

About 10% of uninfected hemodialysis patients, are at risk of acquiring new HCV infection, following transplantation, particularly those receiving more than one kidney or more than five units of blood. Of about 50 % of the organ recipient patients, from donors who were positive for antibody to HCV developed hepatitis after organ transplantation. Hence antibody positivity to HCV is now an exclusion criterion for organ donation.

The transmission of HCV can also occur through non percutaneous mode which includes, transmission that occurs between sexual partners and the transmission from mother to the offspring. The available evidences suggest that the non percutaneous transmission routes, including sexual route, is rare and inefficient when compared to transmission of hepatitis B virus. The transmission of HCV through sexual route is not well defined.

In prospective studies conducted studying the rate of transmission of HCV in sexual partners of infected patients, the transmission rate seemed to be less than 1% per year, whereas there was much more transmission of HIV and HBV, which indicates that sexual transmission is an important route of HIV and HBV acquisition and not of HCV. The spread of HCV through heterosexual contact is reported from Thailand, Argentina and Egypt. HCV infection through homosexual contact has been reported from European countries.

The risk of acquiring HCV infection in the spouse of an infected patient, in a long term monogamous relationship is about 4 % on an average. The pattern of sexual relationship also plays a significant role in determining the transmission of HCV.

The anti-HCV prevalence was about 1 to 10 % in the sexual partners of index patients, without high risk behaviors like promiscuity or injecting drug use, whereas it was around 11 to 27 % in partners of patients with high risk behavior. Studies also suggest that the sexual transmission of HCV is more efficient from male to female when compared to female to male. Studies show that, presence of genital ulcer disease seems to be associated with increased sexual transmission of HCV.

Men who are involved in homosexual practice have an increased rate of anti-HCV positivity when compared to those who have a monogamous heterosexual contact. Multiple sexual exposure with multiple partners and injecting drug use adds to the risk of increased acquisition. A study conducted in homosexual men, the risk factors for sexual transmission of HCV were anal receptive intercourse, having a sexual partner with injecting drug use, history of genital herpes, and seropositivity for HIV.

There is a steady increase in acute HCV infection in HIV seropositive homosexual men, during the last ten years, in various parts of the world , and this is attributed to traumatic anal sex, presence of other genital ulcer disease like syphilis, herpes and lymphogranuloma venereum, and recreational drug usage.

The sexual partners of injecting drug users, heterosexuals with multiple partners, prostitutes and their clients are all at an increased risk of acquiring the disease. The coinfection of HIV and HCV increases the sexual transmission of HCV suggesting that HIV may act as a cofactor in HCV transmission. The transmission rate of HCV is 5 times increased when HIV is also transmitted. As a result of immunosuppression in case of HIV coinfection, there is an enhanced viremia, and hence the sexual transmission of HCV is more profound, indicating that viral load is also a very important factor in deciding the rate of transmission.

The significant immunological changes in HIV coinfection and presence of mucosal defects facilitate the acquisition of HCV. Since the cell mediated immunity is defective in HIV, HCV clearance is reduced and increased loads of HCV is found in serum and semen. In HCV/HIV coinfection, the course of liver disease is more rapid and there is twice increased risk of liver cirrhosis when compared to patients with HCV infection alone.[15]

In a study conducted in slums of urban Chennai, regarding the sexual exposure and HCV infection, HCV infected patients gave a history of having suffered from genital ulcer in the past, indicating the significance of presence of

ulcerations in the enhanced disease transmission. The rate of perinatal transmission of HCV is about 2 to 8 % which is very low when compared to perinatal transmission of hepatitis B. In case of HIV/HCV coinfection , the transmission rate is increased to 36 to 44 %. The greater risk of HCV acquisition in the fetus is associated with the higher titres in the mother. Acute HCV infection presents with asymptomatic elevation in the levels of liver enzymes. Constitutional symptoms like anorexia, loss of weight, pain abdomen, myalgia and mild jaundice can occur. All the symptoms usually resolve in a period of about three months.

The occurrence of fulminant hepatic failure is very uncommon in HCV infection. As early as 1 week after exposure, the HCV-RNA, can be detected by PCR in acute hepatitis C infection. The antibodies against HCV can be detected about 4 to 8 weeks after exposure, though it may take about more than three months to appear. In about 60 to 70%, the risk of chronic infection is present. In chronic HCV infection, fatigue is the commonest symptom, which is followed by right sided upper abdominal pain.

Hepatomegaly and splenomegaly is present in patients who have developed liver cirrhosis. Enzyme elevation is widely variable in the course of the disease. Histologically chronic hepatitis C is associated with characteristically, more lobular and degenerative changes, macrovesicular steatosis, eosinophilic granules, sinusoidal cell activation and bile ductal lesions. Chronic hepatitis C has a slow progression, requiring 1 to 2 decades post infection, for minimal deterioration to

occur. But the survival rate is poor in patients who have developed cirrhosis, which is only 50% survival in more than 5 years. The cofactors implicated in the progression of HCV includes alcohol intake, male sex, age above 45 years, ethnicity, the immune status of the host and the presence of HIV coinfection.

The extrahepatic manifestations of chronic HCV includes presence of autoantibodies such as antinuclear, anti thyroid and anti smooth muscle antibodies, essential mixed cryoglobulinemia, cutaneous leucocytoclastic vasculitis, cryoglobulinemic renal disease, arthralgia, porphyria cutanea tarda, lichen planus, polyarteritis nodosa, urticaria, prurigo, erythema nodosum and erythema multiforme, non hodgkins B cell lymphoma and autoimmune thrombocytopenia purpura.

Detection of antibodies against HCV antigen, serum HCV RNA, and HCV genotyping are employed in the diagnosis of HCV infection. Pegylated interferon alpha is used in the treatment of acute hepatitis C infection and in chronic infection, weekly pegylated interferon alpha along with daily ribavirin is used in treatment.

Hepatitis B

Hepatitis B is caused by a DNA virus, the Hepatitis B Virus, a prototype of hepatotropic DNA viruses (hepadnaviruses). The infectious virion is a spherical and double shelled structure, of 42 nanometer diameter size, known as the Dane particle. The outer shell is the surface antigen-HBsAg, surrounding the inner core protein-the core antigen-HBcAg containing nucleocapsid. The HBV genome is relatively stable. Seven genotypes are described-A to G, based on the divergence in nucleotide sequence of 8 % or more.

The genotypes have a different geographic distribution. Genotype D is common in India. The pathogenesis and the treatment response to antiviral agents vary between the different genotypes. In different parts of the world , the carrier rate ranges from 0.1% to 20%. In India it is between 1.1% to 12.2% in the general population.

The transmission of hepatitis B virus is mainly through three ways- vertical transmission, sexual route and parenteral transmission. A source, an effective mode of spread and a susceptible host are all essential for the transmission to occur. The blood and blood products of chronic carriers of HBV is the source of HBV infection. A single and a minute exposure is sufficient to transmit the disease, since the hepatitis B virus is present in very large amounts in the blood.

The viral load in the blood correlates with the infectivity of the sample. The presence of HBeAg is significant, because when present along with HBsAg, the viral load is very high and hence a minor needle stick injury is sufficient for disease transmission. Transmission can also occur through HBsAg negative blood transfusion during the window period of HBV infection.

The concentration of HBsAg in urine, feces, breast milk, sweat, tears, vaginal secretions and cerebrospinal fluid is very low and hence the secretions have not proven to be infectious. Infection has been reported to be transmitted through semen, but the amount of virus in semen is around 100 to 1000 times less than that in the blood.

Therefore the major risks in parenteral transfusion are after blood transfusion, untreated plasma products exposure, needle stick injuries, use of unsterilized instruments, tattooing, acupuncture and dentistry. Injecting drug use is also a proven source of disease transmission. In the past when blood and blood products were used without screening, hemophiliacs, who were frequently transfused with factor VIII concentrates, had a higher incidence of hepatitis B infection.

In areas with a low to intermediate level of prevalence of infection, sexual route is the most important way of HBV transmission. The manner of sexual transmission of infection has not been clearly defined.

HBV antigen can be detected in semen, and the breach in the skin or the mucous membrane increases the rate of transmission. Sexual activity has been incriminated as the source of transmission in anal intercourse, particularly in the receptive partners. In case of heterosexuals, the spread occurs through minute lacerations in the penis or the vagina and when HBV is present in the semen or the vaginal secretions.

The risk factors for increased transmission in heterosexuals include number of sexual partners, duration of sexual activity, reactive serology for syphilis, and a history of other sexually transmitted infections. Risk factors for viral acquisition in homosexual population includes, having multiple sexual partners, receptive anal intercourse, and the duration of sexual activity. Commercial sex workers and the clients, and partners of injecting drug users are at increased risk of acquiring HBV infection.

Active viral replication with high HBV-DNA/HBeAg positivity facilitates the sexual transmission of the infection. Condom usage appears to decrease the risk of sexual transmission. Among the sexual contacts of men who suffered from hemophilia or injecting drug users, the higher rates of antibodies were found against hepatitis B than HIV or HCV indicating that HBV may be more rapidly spread by sexual contact than HIV and HCV.[85]

In areas of high prevalence of HBV, perinatal transmission is a very significant mode of transmission. High plasma viral load, especially when it is more than 2×10000 IU/ml is associated with an increased rate of transmission. The high viral load and HBeAg positivity in the mother is more likely to accelerate the transmission of the disease. The babies infected are asymptomatic and they have about 90% chances of developing a chronic infection which is more in comparison to adults where there is only 5 to 10 % chance of developing a chronic infection.

The infant acquires the infection through inoculation of maternal blood or liquor that occurs during the passage through the vaginal canal, rather than the intrauterine transmission.[85] After the exposure to the virus occurs, the virus is transmitted through blood to the liver. The virus gets attached to the hepatocyte. The uptake of HBV into hepatocyte is mediated by sialoglycoprotein receptors on the hepatocyte, possibly through endocytosis.

When the virus enters the hepatocyte, the nucleocapsid is transferred to the nucleus, followed by translation and reverse transcription and finally assembly of mature virions. During the process of the viral replication, some of the HBV-DNA gets integrated into the host chromosomal DNA. This integrated HBV-DNA is implicated in the development of hepatocellular carcinoma after many years.

In acute hepatitis, maximal HBV replication occurs before the maximal cellular injury, indicating that the disease might represent the immunologically mediated lysis of the infected hepatocytes. Even at the maximal concentrations of virus in the serum and liver, liver disease is very minimal, with little or no evidence of hepatocyte necrosis on biopsy, which suggests that the virus is not cytopathic.

An efficient HLA class 1 and class 2 restricted T cell response to viral proteins clears virus in acute hepatitis B . In chronic infection, there is a weak HLA class 2 restricted T cell response, which is insufficient for clearing the replicating virus. Patients with immunosupression , for example those on hemodialysis, post renal transplant recipients, patients suffering from leukemia or leprosy and patients with HIV coinfection are more prone for chronic infection. Clinically, the manifestations of hepatitis B infection depend on the factors like age at which the infection occurs, the immunological status of the host, the rate of replication of the Hepatitis B Virus.

A spectrum of manifestations are seen ranging from subclinical hepatitis to anicteric hepatitis followed by icteric hepatitis proceeding to fulminant hepatic failure in acute phase of the infection. The incubation period of acute hepatitis B ranges from 40 to 140 days. The preicteric or the prodromal phase consists of symptoms like anorexia, nausea, vomiting, fatigue, fever of low grade, malaise and myalgia. Few patients have a serum sickness like syndrome and a generalized

maculopapular rash or urticaria. This phase lasts for about 3 to 7 days, followed by icteric phase, where jaundice occurs and the constitutional symptoms decrease. This phase may last upto twelve weeks, followed by a convalescent phase, where there is a resolution of jaundice and disappearance of other symptoms and improvement in appetite. The last symptom to disappear is fatigue.

The clinical examination in acute hepatitis shows icterus, fever of low grade and tender and soft hepatic enlargement. There is a 10 to 50 fold elevation of levels of alanine aminotransferase and aspartate aminotransferase. Serum bilirubin levels and prothrombin time are the markers of prognosis. The persistently elevated levels of ALT for a period of more than 6 months duration are suggestive of chronic liver injury.

Liver biopsy reveals disarray of lobular architecture, hepatocytes showing acidophilic degeneration, focal lobular necrosis, lymphocytic and macrophage infilteration of the parenchyma, kupffer cell hypertrophy and hyperplasia, bile ductules disruption and cholestasis. HBsAg is the first serological marker that is detected in the serum, which appears during the incubation period and lasts upto 5 months or longer.

The first antibody to appear is IgM antiHBc and persists upto 12 months. IgG antiHBc also appears early and persists for life.[85] In the chronic phase of the infection, the spectrum ranges from a carrier state which is asymptomatic to chronic hepatitis, followed by cirrhosis, ultimately proceeding to hepatocellular

carcinoma. As the age at which the infection is acquired, increases,the probability of developing chronicity, decreases. Chronic infection is more common in men. In a majority of patients, the onset of infection is mild or totally asymptomatic or may have nonspecific symptoms like intermittent fatigue which worsens with exertion or the patients may have anorexia, nausea, jaundice, low grade fever and weight loss. When the patient develops cirrhosis, there is weakness, weight loss, wasting, edema, abdominal swelling, jaundice, encephalopathy and variceal bleeding.

Few may present with extrahepatic manifestations. Rarely few patients may present with hepatocellular carcinoma. Clinically edema, abdominal swelling, spider angiomas, splenomegaly, distended veins over abdomen and ascites is seen in patients with cirrhosis. Esophageal varices may be seen on endoscopy. There may a 5 to 8 fold rise in aminotransferase levels with ALT higher than AST and AST:ALT ratio lesser than1.

With the increasing liver dysfunction, prothrombin time and serum bilirubin increases and serum albumin decreases all pointing towards a poor prognosis. Few patients may develop a complicated clinical course characterized by fulminant hepatitis, post hepatitis syndrome, relapses and chronic hepatitis. Hepatocellular carcinoma occurs after decades of chronic HBV infection. The patients present with constitutional symptoms, abdominal mass and a decompensated liver disease.

In acute hepatitis B, lamivudine or entacavir are indicated in cases of impending acute liver failure. In case of chronic hepatitis B, pegylated interferon alpha, lamivudine, adefovir, entacavir, telbivudine, tenofovir and emtricitabine are approved for treatment. The aim is to achieve a consistent suppression of viral replication and disease remission.

Hepatitis B, Hepatitis C and HIV coinfection:

The routes of transmission and risk behaviors for HBV and HCV are similar to those of HIV transmission. In a study conducted in patients attending STD outpatient departments of district hospitals of Northern part of India, it was found that HBV/HIV coinfection in 0.2%, and no HIV/HCV coinfection.[88]

In a study conducted in STD outpatient department at Jipmer, Pondicherry, documents a significant role of sexual route of transmission of HIV,HCV and HBV, HIV having the highest risk followed by HCV and HBV. An increased incidence of HCV positivity was found in patients with genital ulcer disease and HIV infection.

The male patients with any one of the markers for either anti HBV/anti HCV/antiHIV were more likely to have each one of other markers.[89] In a prospective study conducted in S.N. Medical College, Uttar Pradesh, India, on comparing the HIV and HBV coinfections, the coinfection rates were higher in people infected with HIV.[90]

Persons infected with HIV are at higher risk of acquiring HBV. The coinfection of HBV with HIV worsens the prognosis of the liver disease, more prone for chronic HBV infection, and also reduces the rate of response to interferons. In people infected with HIV and with immunosupression, reactivation or reinfection with HBV is common. All patients with HBV infection, should be screened for coexistent HIV infection, to decide on treatment options and to prevent the emergence of drug resistance.

The only drugs that can be used as monotherapy in HBV/HIV coinfection are adefovir and pegylated interferon.[85] Hepatitis C virus causes a rapid progression of HIV disease, and HIV coinfection impairs the treatment response of HCV. Early diagnosis and appropriate treatment of genital ulcers is essential in the prevention of spread of sexually transmitted diseases.

Aims and Objectives

AIM OF THE STUDY

1. To study about the coincidence of Retroviral disease (HIV), Hepatitis B and Hepatitis C in male patients with genital ulcer attending the STD O.P. Institute of Venereology, Government General Hospital.
2. To study about the association of clinico epidemiological factors with Genital Ulcer Disease.
3. To study about the sexual orientation in patients with Genital Ulcer Disease with the coincidence of HIV, Hepatitis B and Hepatitis C.
4. To determine the commonest causes of Genital Ulcer Disease, coinciding with HIV, Hepatitis B and Hepatitis C.

Materials and Methods

MATERIALS AND METHODS

STUDY DESIGN

Prospective study

SAMPLE

The study population comprised a total of 100 male patients with genital ulcer attending the STD O.P. Institute of Venereology, Government General Hospital, Chennai from 1st November 2016 to 31stAugust 2017.

METHODS

The patients were interviewed regarding age, educational status, occupation, marital status, presenting complaints, sexual history, past history of venereal disease and condom usage.

Counseling was given to all patients regarding sexually transmitted disease, protective sexual practices, prompt treatment and regular follow up. Pre and post test counseling was given.

Complete general examination and genital examination was done for all the patients after recording a detailed history. Dark field, tzanck smear, gram stain, tissue smear were done in all patients from the genital ulcer to rule out syphilis, herpes, chancroid, donovanosis respectively. In addition, wet mount examination was done for trichomoniasis and candidiasis.

Serological investigations were done for HIV, HBSAg, anti HCV antibodies, VDRL and TPHA tests were done to rule out syphilis.

Specific investigations like cerebrospinal fluid analysis and CD4 cell count were done in patients, if needed.

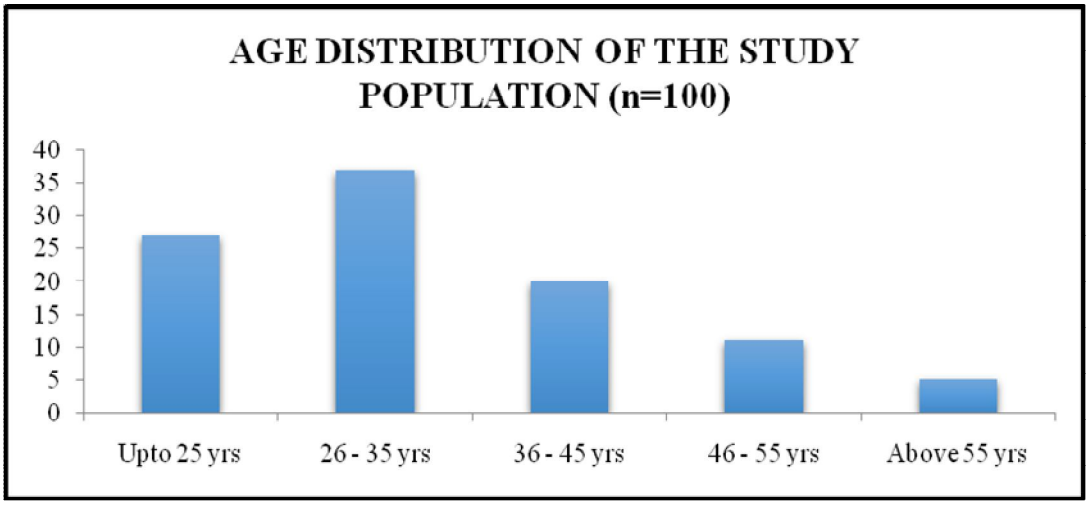
Routine baseline laboratory analysis like complete blood count and urine routine analysis were done for all patients. Liver and renal function tests, chest X ray and ECG were done in the needed patients.

Observation & Results

OBSERVATION AND RESULTS

AGE DISTRIBUTION OF THE STUDY POPULATION (n=100)

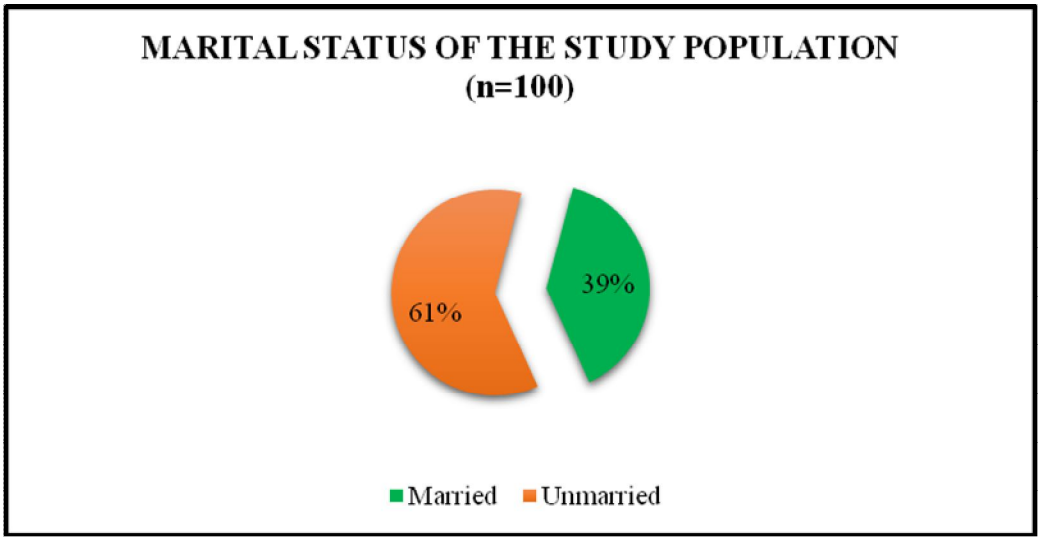
AGE DISTRIBUTION	FREQUENCY	PERCENTAGE
Upto 25 yrs	27	27.0%
26 - 35 yrs	37	37.0%
36 - 45 yrs	20	20.0%
46 - 55 yrs	11	11.0%
Above 55 yrs	5	5.0%
Total	100	100.0%



The most common age group affected with the genital ulcer disease in the study population was between 26 - 35 years, and they were 37 in number (37%), followed by patients of age less than 25 yrs, who were 27 in number (27%) and patients in the age group of 36 - 45 yrs, 20 in number (20%) patients of 46 - 55 yrs, 11 in number (11%), and patients above 55 yrs constituted the least, and were 5 in number (5%).

MARITAL STATUS OF THE STUDY POPULATION (n=100)

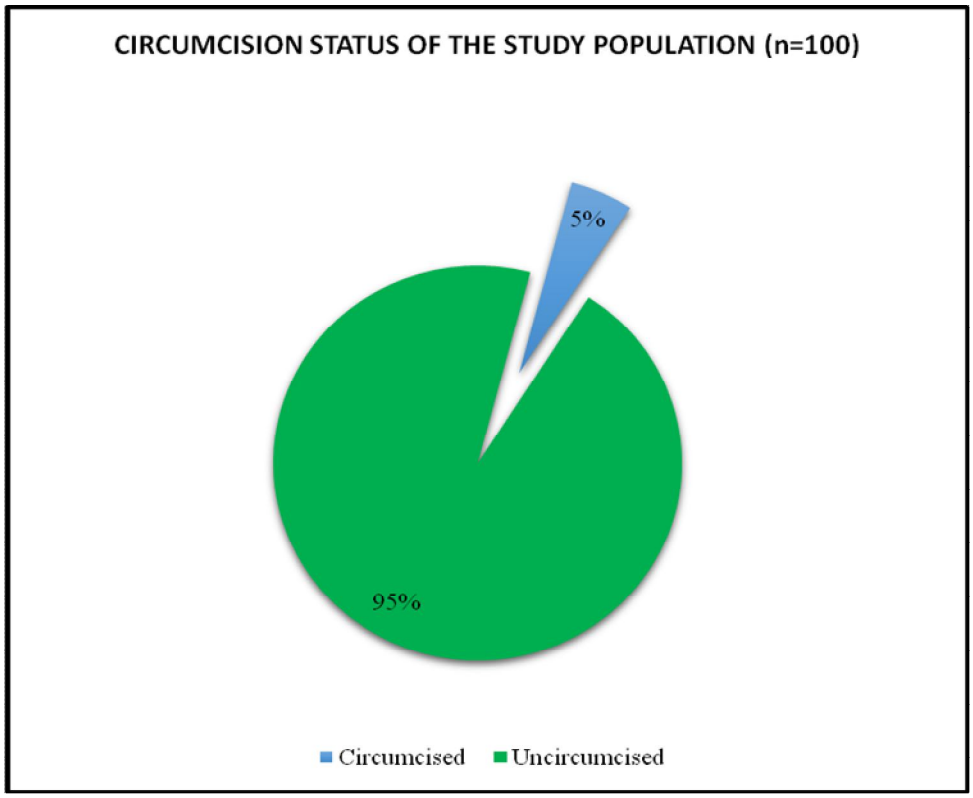
MARITAL STATUS	FREQUENCY	PERCENTAGE
Unmarried	61	61.0 %
Married	39	39.0 %
Total	100	100.0 %



Among the 100 patients in the study population (n=100) unmarried patients were more commonly affected with the genital ulcer disease, constituting about 61 in number (61%) as against 39 patients (39%) who were married.

CIRCUMCISION STATUS OF THE STUDY POPULATION (n=100)

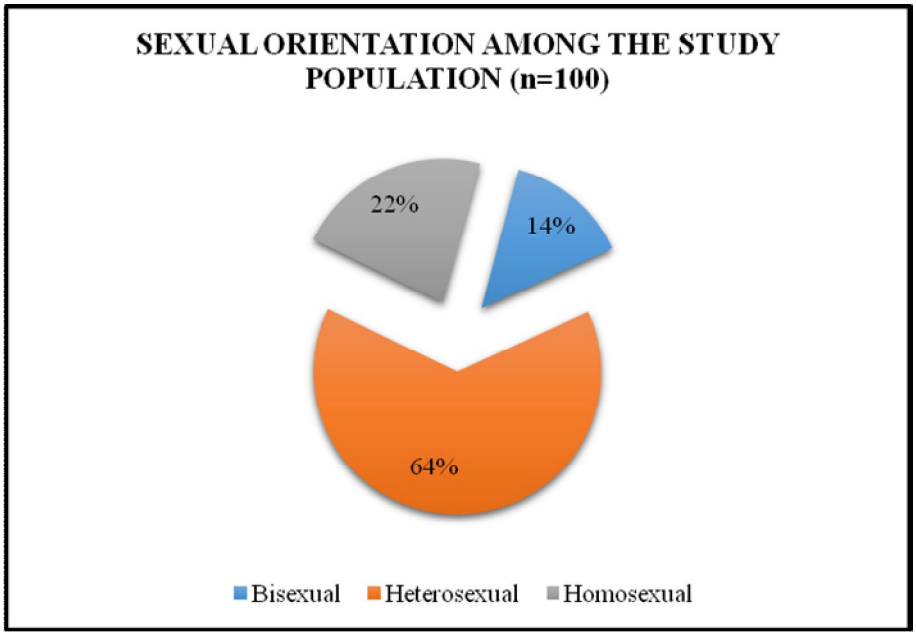
CIRCUMCISION STATUS	FREQUENCY	PERCENTAGE.
Uncircumcised	95	95.0 %
Circumcised	5	5.0 %
Total	100	100.0 %



Among the 100 patients in the study population (n=100) presented with genital ulcer disease, 95 patients (95%) were uncircumcised and 5 patients (5%) were circumcised.

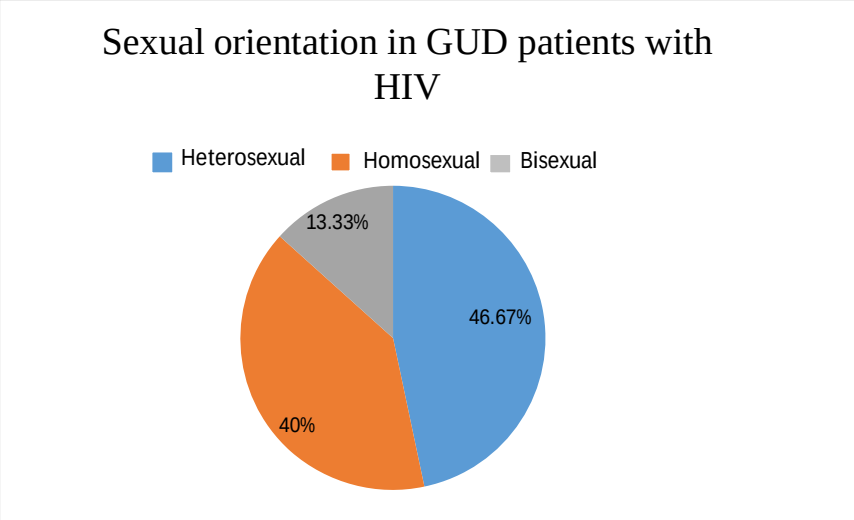
SEXUAL ORIENTATION AMONG THE STUDY POPULATION (n=100)

SEXUAL ORIENTATION	FREQUENCY	PERCENTAGE
Heterosexual	64	64.0 %
Homosexual	22	22.0 %
Bisexual	14	14.0 %
Total	100	100.0 %



Among the 100 patients in the study population (n=100), affected with the genital ulcer disease, heterosexual patients were the most common , and were about 64 in number (64%), followed by homosexual patients, 22 in number (22%) and the least being bisexual patients, 14 in number (14%).

SEXUAL ORIENTATION IN PATIENTS WITH GENITAL ULCER DISEASE (GUD) POSITIVE FOR RETROVIRAL DISEASE (HIV)

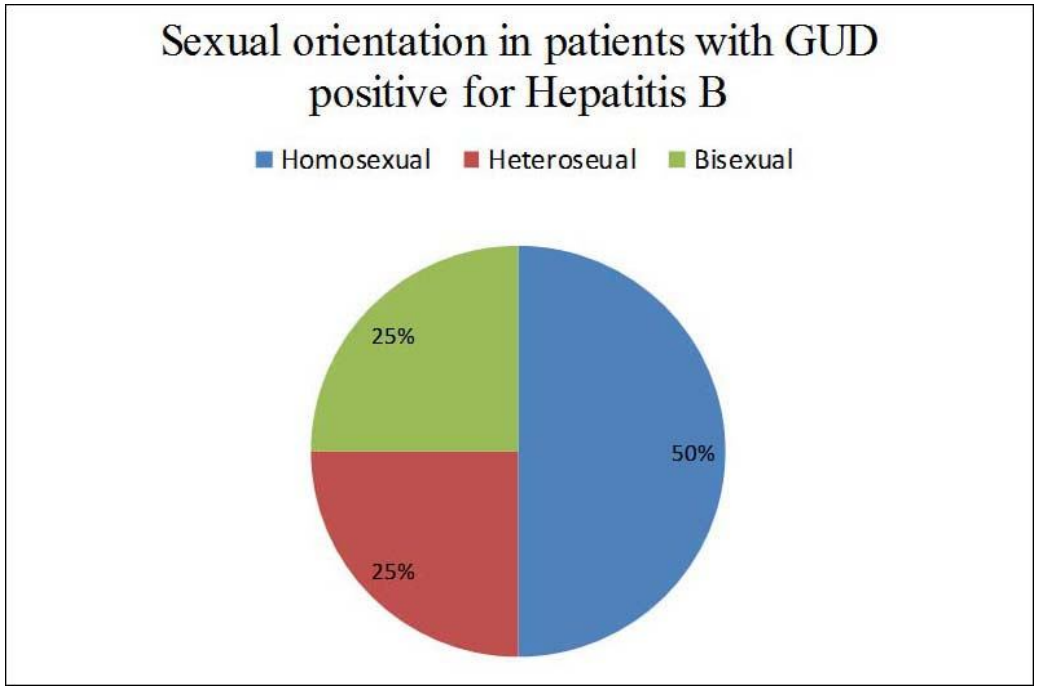


SEXUAL ORIENTATION	FREQUENCY	PERCENTAGE
Heterosexual	7	46.67%
Homosexual	6	40%
Bisexual	2	13.33%
Total	15	100%

Among the 15 patients in the study population, who were found to be positive for Retroviral disease, 7 patients (46.67%) were found to have heterosexual contact, 6 patients (40%) were found to have homosexual contact and 2 patients (13.33%) were found to have bisexual contact.

SEXUAL ORIENTATION IN PATIENTS WITH GUD AND POSITIVE FOR HEPATITIS B

SEXUAL ORIENTATION	FREQUENCY	PERCENTAGE
Homosexual	2	50%
Heterosexual	1	25%
Bisexual	1	25%
Total	4	100%

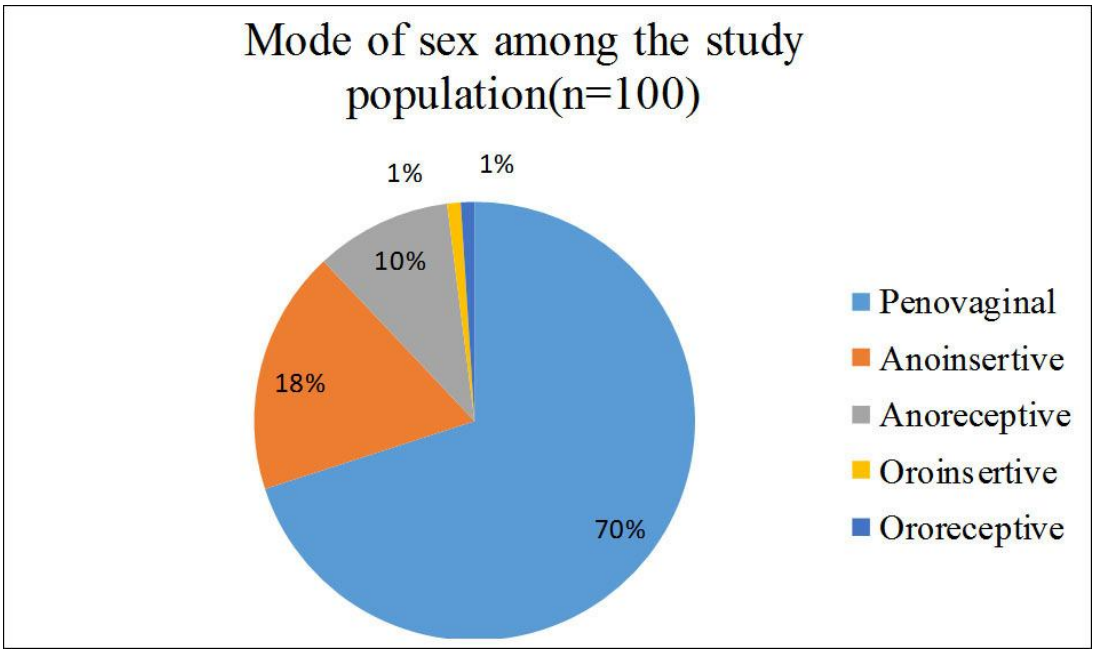


Among the 4 patients who were positive for Hepatitis B in the study population, 2 patients (50%) were found to have homosexual contact, 1 patient (25%) had heterosexual contact and 1 patient (25%) was found to have bisexual contact.

The one patient (1%) positive for Hepatitis C in the study population of 100 patients (n=100) with genital ulcer, was found to have heterosexual contact.

MODE OF SEX AMONG THE STUDY POPULATION (n=100)

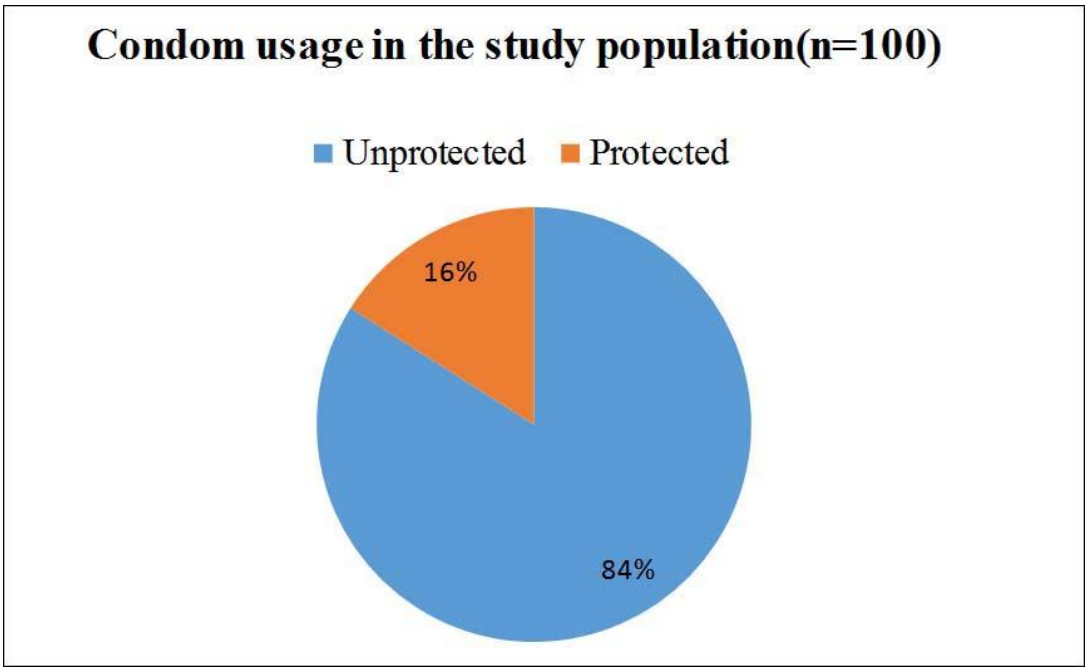
MODE OF SEX	FREQUENCY	PERCENTAGE
Penovaginal	70	70.0 %
Anoinsertive	18	18.0%
Anoreceptive	10	10.0 %
Oroinsertive	1	1.0 %
Ororeceptive	1	1.0 %
Total	100	100.0 %



In the study population of 100 patients (n=100), presented with genital ulcer disease, penovaginal intercourse was the most common mode of sex, found in 70 patients (70%), followed by anoinsertive intercourse in 18 patients (18%), anoreceptive intercourse in 10 patients (10%), oroinsertive intercourse in 1 patient (1%) and ororeceptive intercourse in 1 patient (1%).

CONDOM USAGE IN THE STUDY POPULATION (n=100)

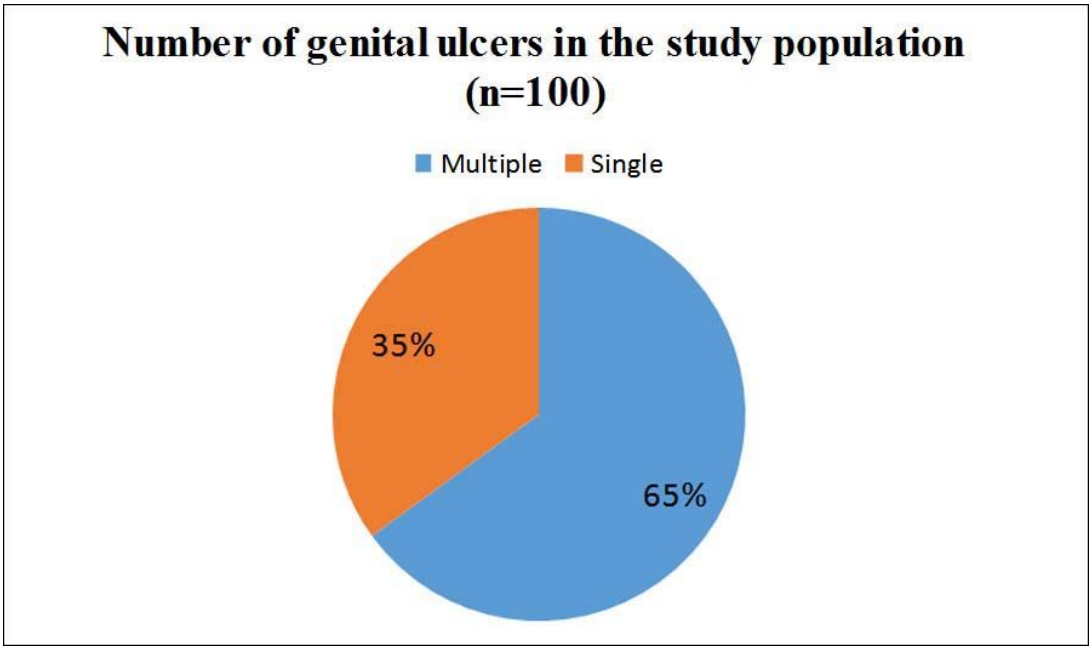
CONDOM USAGE	FREQUENCY	PERCENTAGE
Unprotected	84	84.0 %
Protected	16	16.0 %
Total	100	100.0%



Among the 100 patients in the study population (n=100), presented with genital ulcer disease, 84 patients (84%) followed unprotected sexual intercourse and formed a larger group than 16 patients (16%)who followed protective sexual practices.

NUMBER OF GENITAL ULCERS IN THE STUDY POPULATION (n=100)

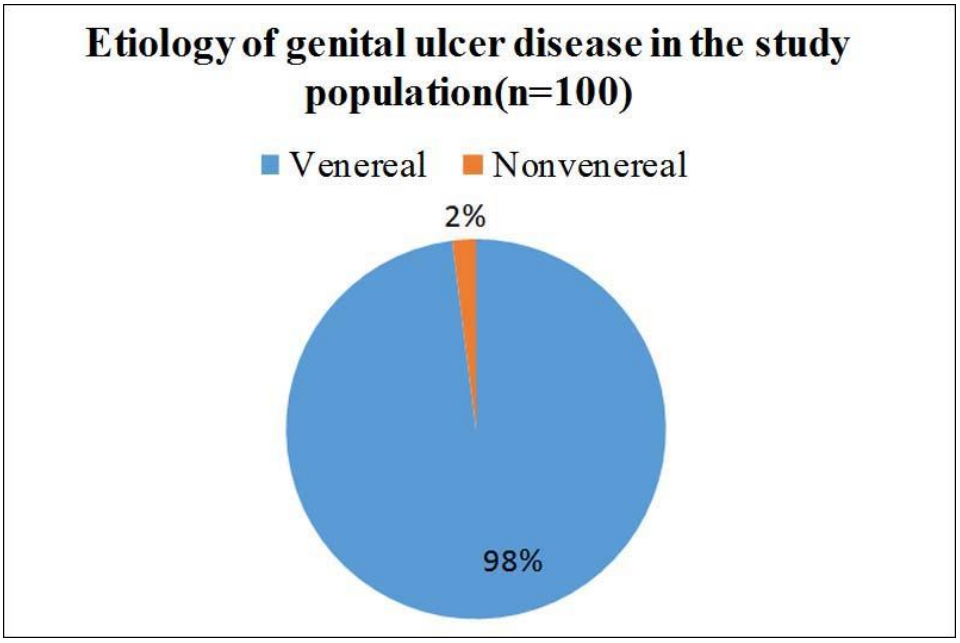
NUMBER OF GENITAL ULCERS	FREQUENCY	PERCENTAGE
Multiple	65	65.0%
Single	35	35.0%
Total	100	100.0%



Among the study population of 100 patients (n=100), 65 patients (65%) had multiple ulcers and 35 patients (35%) had single ulcer.

ETIOLOGY OF GENITAL ULCER DISEASE IN THE STUDY POPULATION (n=100)

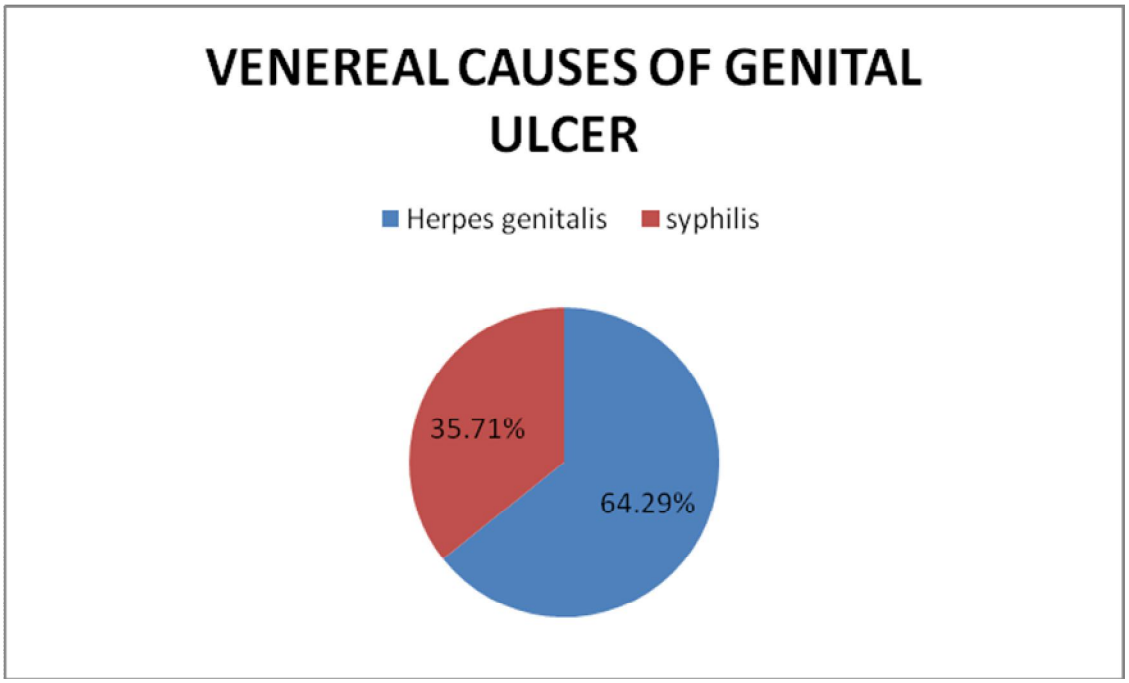
ETIOLOGY	FREQUENCY	PERCENTAGE
Venereal	98	98.0%
Non venereal	2	2.0%
Total	100	100.0%



Among the 100 patients in the study population,(n=100), who presented with genital ulcer disease, 98 patients (98%) had ulcer due to venereal etiology and 2 patients (2%) had ulcer due to non venereal cause.

VENEREAL CAUSES OF GENITAL ULCER

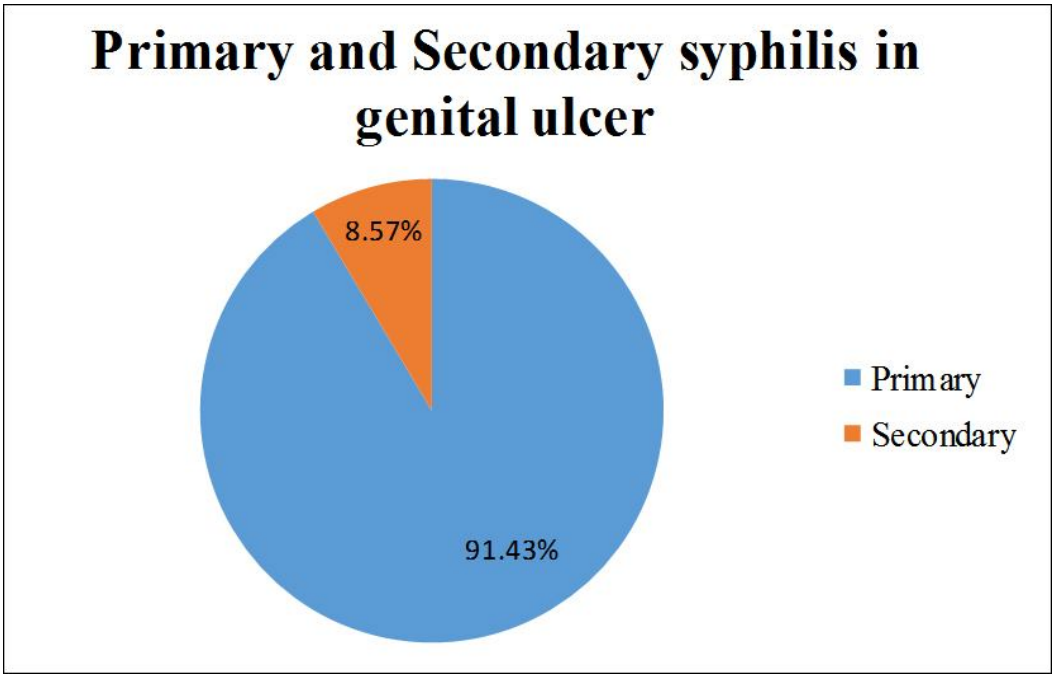
VENEREAL CAUSE	FREQUENCY	PERCENTAGE
Herpes genitalis	63	64.29%
Syphilis	35	35.71%
Total	98	100.0%



Among 98 patients with genital ulcer due to venereal causes, 63 patients (64.29%) had ulcer due to herpes genitalis and 35 patients (35.71%) were found to have ulcer due to syphilis.

INCIDENCE OF PRIMARY AND SECONDARY SYPHILIS IN GENITAL ULCER

SYPHILIS	FREQUENCY	PERCENTAGE
Primary	32	91.43%
Secondary	3	8.57%
Total	35	100.0%



Among the 35 patients who had syphilis along with genital ulcer, 32 patients (91.43%) had ulcer due to primary syphilis and 3 patients(8.57%) had features of primary and secondary syphilis.

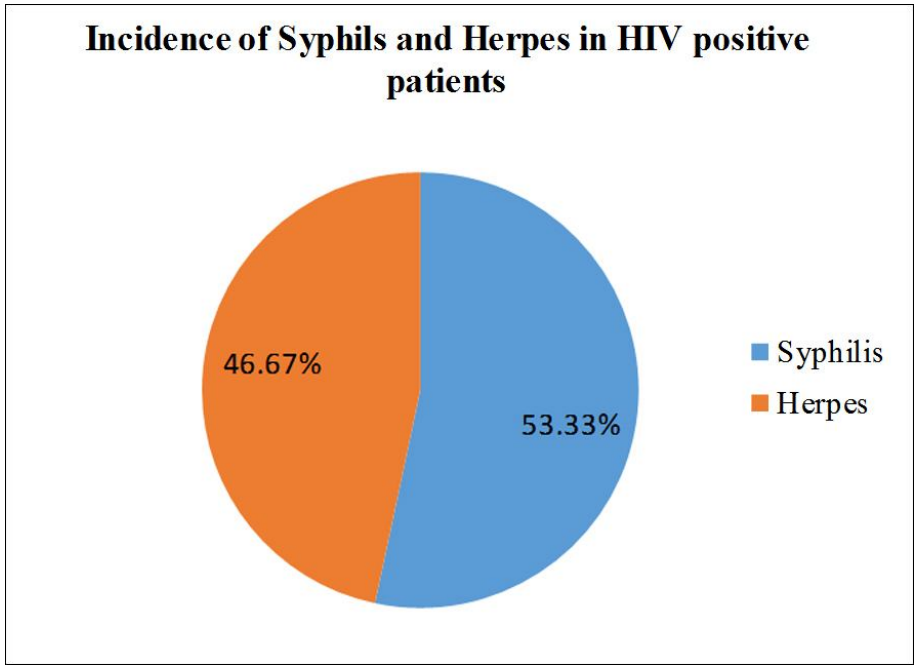
INCIDENCE OF HIV, HEPATITIS B AND HEPATITIS C IN THE STUDY POPULATION

DISEASE	FREQUENCY	PERCENTAGE
HIV	15	15.0%
HEPATITIS B	4	4.0%
HEPATITIS C	1	1.0%

Among the 100 patients in the study population (n=100), who presented with genital ulcer, 15 patients (15%) were found to be positive for HIV, 4 patients (4%) positive for Hepatitis B and 1 patient (1%) was positive for Hepatitis C.

INCIDENCE OF SYPHILIS AND HERPES GENITALIS IN HIV POSITIVE PATIENTS

SYPHILIS/HERPES	FREQUENCY	PERCENTAGE
SYPHILIS	8	53.33%
HERPES GENITALIS	7	46.67%
TOTAL	15	100.0%

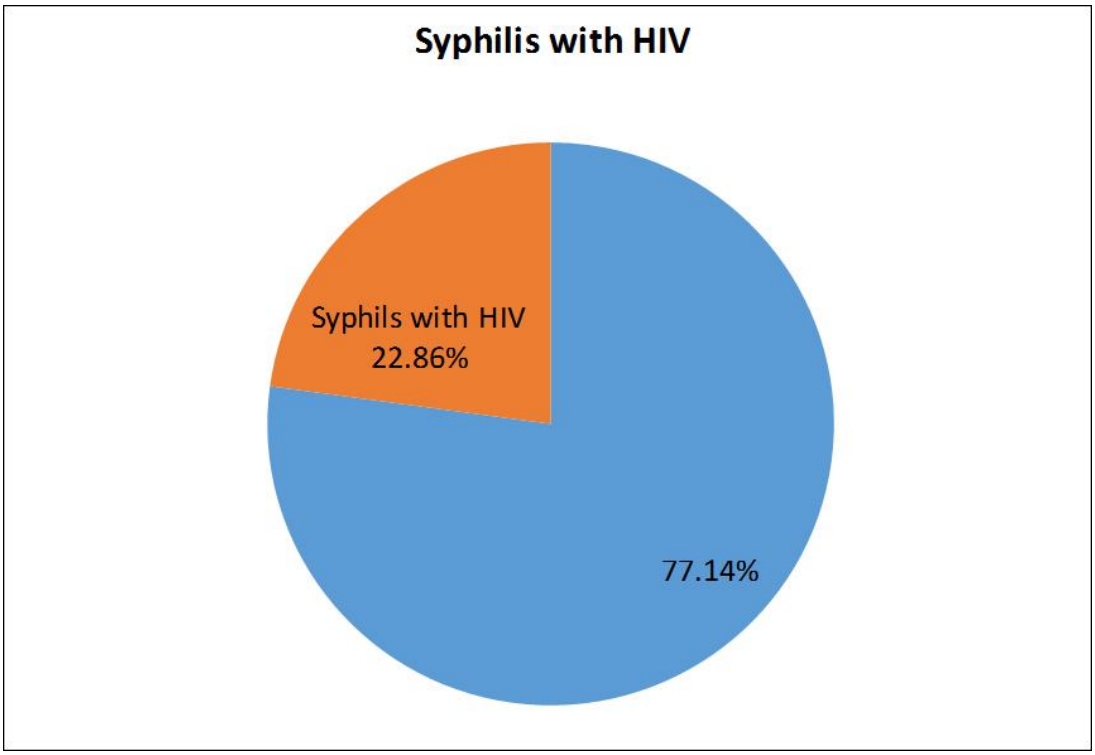


Among the 15 patients with GUD in the study population, who were positive for HIV, 8 patients (53.33%) were found to have ulcer due to syphilis and 7 patients (46.67%) had ulcer due to herpes genitalis.

None of the patients with genital ulcer due to non venereal cause, had positivity for HIV.

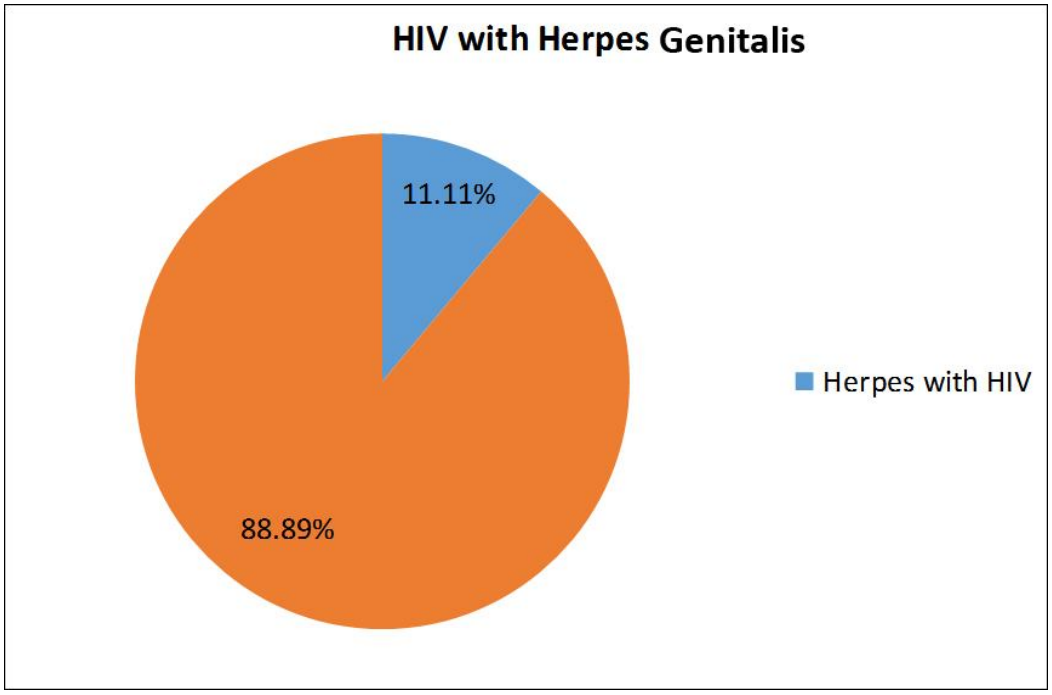
INCIDENCE OF RETROVIRAL DISEASE IN PATIENTS WITH GENITAL ULCER DUE TO SYPHILIS

In the study population of 100 patients, 35 patients (35%) had GUD due to syphilis , 8 patients (22.86%) were found to be positive for Retroviral disease. Rest of the 27 patients(77.14%) were HIV negative. Among the 8 HIV positive patients, 7 patients (87.5%) had primary syphilis and 1 patient(12.5%) had secondary syphilis.



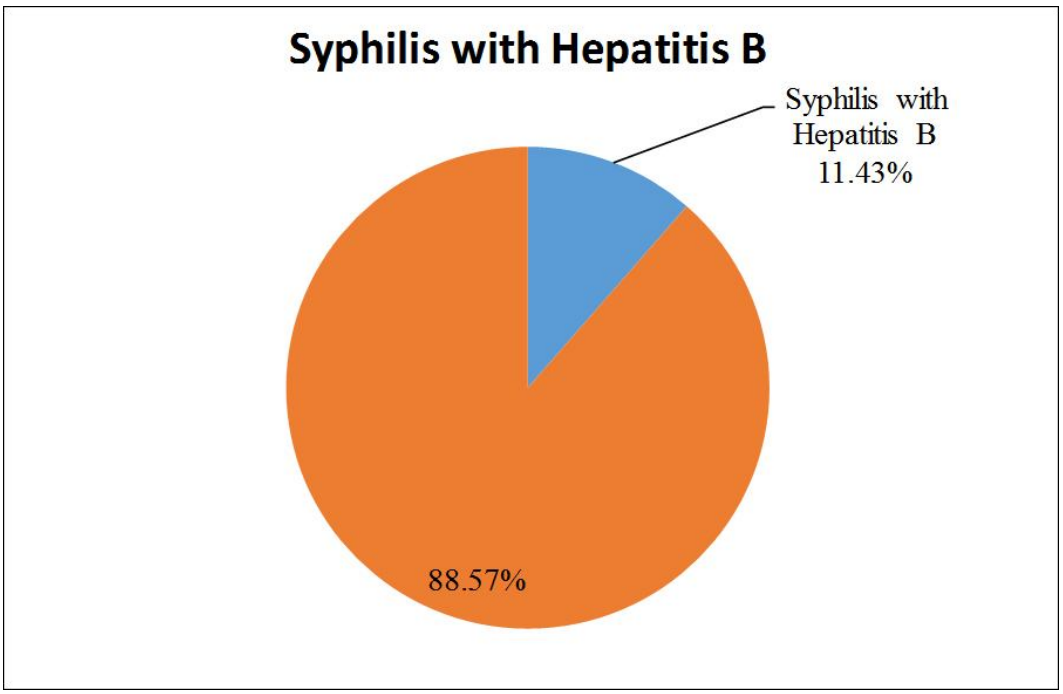
INCIDENCE OF RETROVIRAL DISEASE IN PATIENTS WITH GENITAL ULCER DUE TO HERPES GENITALIS

Among the 100 patients in the study population, 63 patients had ulcer due to herpes genitalis. Among the 63 patients , 7 patients (11.11%) were found to be positive for Retroviral disease and rest of the 56 patients (88.89%) were negative for Retroviral disease .



INCIDENCE OF HEPATITIS B IN PATIENTS WITH GENITAL ULCER DUE TO SYPHILIS

Among the 100 patients who formed the study population(n=100), 35 patients had genital ulcer due to syphilis. Among the 35 patients , 4 patients (11.43%) were found to be positive for Hepatitis B. Rest of the 31 patients(88.57%) were negative for Hepatitis B.



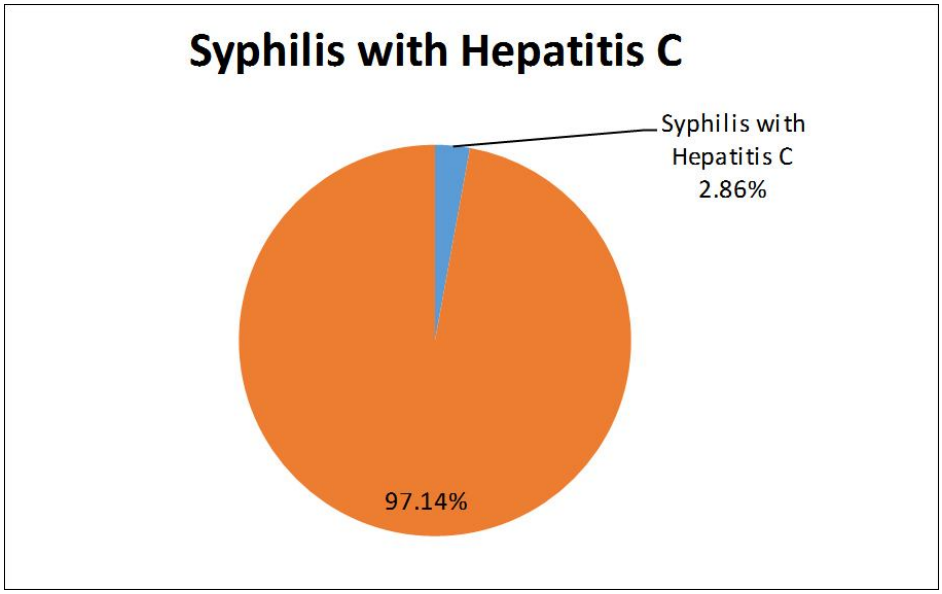
Among the 100 patients who formed the study population (n=100), 35 patients had genital ulcer due to syphilis. Among the 35 patients, 3 patients (8.57%) had secondary syphilis . Among the 3 patients, one patient (33.33%) was positive for Hepatitis B**.**

Among the study population of 100 patients with genital ulcer disease, 63 patients had ulcer due to herpes. None of the 63 patients had positivity for Hepatitis B.

Among the 100 patients(n=100) with genital ulcer disease in our study, 2 patients(2%) had ulcer due to non venereal cause. Neither of the 2 patients had positivity for Hepatitis B.

INCIDENCE OF HEPATITIS C IN PATIENTS WITH GENITAL ULCER DUE TO SYPHILIS

Among the 100 patients (n=100) who formed the study population, 35 patients had genital ulcer due to syphilis. Among the 35 patients (n=35), 1 patient (2.86%) was found to be positive for Hepatitis C and rest of the 34 patients (97.14%) were negative for Hepatitis C.



Among the study population of 100 patients with genital ulcer disease, 63 patients had ulcer due to herpes. None of the 63 patients had positivity for Hepatitis C.

Among 100 patients with genital ulcer disease, 2 patients had ulcer due to non venereal cause. Neither of the 2 patients had positivity for Hepatitis C.

COINCIDENCE OF HIV, HEPATITIS B AND HEPATITIS C IN THE STUDY POPULATION WITH GENITAL ULCER DUE TO SYPHILIS.

Among the 100 patients who presented with genital ulcer, 35 patients had genital ulcer due to syphilis, and among the 35 patients one patient (2.86%) had positivity for all the three viruses- HIV, Hepatitis B and Hepatitis C.

Though Herpes genitalis, constituted the major cause of ulcer disease (63%) among the study population of 100 patients, none of the patients had a coincidence of all three viruses.

Among the 100 patients of the study population with genital ulcer disease, 2 patients (2 %) had ulcer due to non venereal causes. Neither of the two patients showed reactivity for any of the three viruses – HIV, Hepatitis B or Hepatitis C.

COINCIDENCE OF HIV AND HEPATITIS B IN THE STUDY POPULATION (n=100).

Among the 100 patients who formed the study population (n=100), coincidence of Hepatitis B and HIV was seen in 1 patient (1%).

COINCIDENCE OF HIV AND HEPATITIS C IN THE STUDY POPULATION (n=100).

Among the 100 patients who formed the study population (n=100), coincidence of Hepatitis C and HIV was seen in 1 patient (1%).

COINCIDENCE OF HEPATITIS B AND HEPATITIS C IN THE STUDY POPULATION (n=100).

Among the 100 patients who formed the study population (n=100), coincidence of Hepatitis B and Hepatitis C was seen in 1 patient (1%).

COINCIDENCE OF HIV, HEPATITIS B AND HEPATITIS C IN THE STUDY POPULATION (n=100).

Among the 100 patients who formed the study population (n=100), coincidence of HIV, Hepatitis B and Hepatitis C was seen in 1 patient (1%).

A single well defined ulcer of size 0.5*0.5 cm over the glans penis

Single, well defined, ulcer, with indurated base present circumferentially over the prepuce

Single well defined ulcer with indurated base present over the coronal sulcus

Multiple well defined superficial ulcers over the glans penis and prepuce

Discussion

DISCUSSION

The most common age group affected with the genital ulcer disease in the study population was between 26 - 35 years, similar to the age distribution in the study by Sirisha Singh et al., in a Jipmer study[89], and they were 37 in number (37%), followed by patients of age less than 25 yrs, 27 in number (27%) and patients above 55 yrs constituted the least, and were 5 in number (5%).

Unmarried patients (61%) were more commonly affected with the genital ulcer disease, and 39 patients (39%) were married.

Among the 100 patients, 95 patients (95%) were uncircumcised and only 5 patients (5%) were circumcised.

Patients were mostly, heterosexuals forming 64%, followed by 22% homosexual patients and the least formed by bisexual patients, of about (14%). Among the 15 HIV positive patients in the study population, 7 (46.67%) had heterosexual contact, 6 patients (40%) had homosexual contact and 2 patients (13.33%) had bisexual contact.

Among the 4 patients positive for Hepatitis B, 2 patients (50%) had homosexual contact, 1 patient (25%) had heterosexual contact and 1 patient (25%) had bisexual contact.

The one patient (1%) positive for Hepatitis C, was found to have heterosexual contact.

The most common mode of sex was penovaginal intercourse forming 70%, followed by anoinsertive intercourse in 18%, anoreceptive intercourse in 10%, oroinsertive intercourse and ororeceptive intercourse in 1% each.

Most of the patients(84%) with genital ulcer practiced unprotected sexual intercourse and 16% practised protective sex.

About 65% of patients had multiple ulcers and single ulcer was found in 35% patients.

Venereal etiology was found to be the commonest cause of genital ulcer about 98 patients(98%) and 2% patients had ulcer due to non venereal etiology.

Among 98 patients with venereal cause of genital ulcer, 63 patients (64.29%) were found to have herpes genitalis and 35 patients (35.71%) had ulcer due to syphilis.

Among the 35 patients with genital ulcer due to syphilis, 32 patients (91.43%) had primary syphilis and 3 patients (8.57%) had primary and secondary syphilis.

Among the 100 patients in the study population, HIV was positive in 15% patients. This is very high compared to the incidence of 2.9% reported in a study in South Africa by G Paz Bailey[91] et al.,and lower than the incidence of 23.2% reported by Sirisha Singh et al, in a JIPMER study.[89]

In the study population, 4% patients had Hepatitis B which is lower than the incidence of 10 % reported in JIPMER study by Sirisha Singh[89] et al, and lower than the Tungatkar[92] et al., study in Pune in STD patients with the reported incidence of 5.78%.

Among the study population, 1% patients had hepatitis C. This is very low compared to 21.1% incidence reported in JIPMER study by Sirisha Singh[89] reported in the Tungatkar et al., study[92] in Pune in STD patients.

Among the 15 patients who were positive for HIV, 8 patients (53.33%) had ulcer due to syphilis and 7 patients (46.67%) had herpetic ulcers.

Neither of the 2 patients with ulcer due to non venereal etiology, had positivity for HIV.

Among the 35 patients (35%) who had GUD due to syphilis , 8 patients (22.86%) were positive for Retroviral disease. The remaining 27 patients (77.14%) were negative for Retroviral disease. 7 (87.5%) among the 8 HIV positive patients, had primary syphilis and 1 patient(12.5%) had secondary syphilis.

Out of the 63 patients who had ulcer due to herpes genitalis, 7 patients (11.11%) were positive for Retroviral disease and the remaining 56 patients (88.89%) were negative for Retroviral disease.

Among the 35 patients who had genital ulcer due to syphilis, 4 patients (11.43%) had Hepatitis B.

Among the 35 patients who had genital ulcer due to syphilis , 3 patients (8.57%) had secondary syphilis . Among the 3 patients, one patient (33.33%) was positive for Hepatitis B.

Out of the 63 patients with herpetic ulcers, none had positivity for Hepatitis B.

Neither of the 2 patients with non venereal ulcers had positivity for Hepatitis B.

Among the 35 patients with syphilis with genital ulcer, 1 patient (2.86%) had positivity for Hepatitis C.

None among the 63 patients who had herpetic ulcers, showed positivity for Hepatitis C.

Neither of the 2 patients with ulcers due to non venereal etiology had Hepatitis C.

Among the 35 patients with genital ulcer and syphilis, one patient (2.86%) had positivity for HIV, Hepatitis B and Hepatitis C.

Herpes constituted the major cause of genital ulcer disease among the study population of about 63% , yet none had a coincidence of all three viruses- HIV, Hepatitis B and Hepatitis C.

Neither of the two patients with non venereal ulcer showed reactivity for HIV, Hepatitis B or Hepatitis C.

Coincidence of HIV and Hepatitis B was seen in one patient (1%) which is lower than the result of 9 % coinfection reported in a study conducted in South India by Shanmugam Saravanan[93] et al.,and slightly higher than the coinfection reported in Amristar study by Neerja Jindal[94] et al., of 2 %.

Coincidence of HIV and Hepatitis C was found in 1 patient (1%), which is lower than the coinfection of 2.2 % reported in a study conducted in South India by Shanmugam Saravanan[93] et al.,and comparable to the 0.85% reported in an Amristar study by Neerja Jindal[94] et al.,

Coincidence of Hepatitis B and Hepatitis C was present in 1 patient (1%).

Coincidence of HIV, Hepatitis B and Hepatitis C was seen in 1 patient (1%). No coinfection of the three diseases were found in Amristar study[94] by Neerja Jindal et al.,among STD clinic attendees.

Conclusion

CONCLUSION

The Genital ulcers, commonly occurring due to herpes genitalis and syphilis are of public health importance, since there is an increased risk of transmitting and acquiring the coinfections like HIV, Hepatitis B and Hepatitis C. Early screening, prompt and appropriate management of genital ulcers may reduce the further transmission of the coinfection. All the sexual partners of the patients should be thoroughly examined, investigated and promptly treated. In addition, safe sex practices like consistent and correct condom usage should be promoted. Preventive vaccination, as in case of Hepatitis B should b considered in all patients with high risk sexual behavior. In our study, the increased coincidence of HIV and the coinfections like Hepatitis B and Hepatitis C are observed in patients suffering from Syphilis, stressing the significance of mandatory screening for all the three viruses in all patients with genital ulcers and especially in those due to Syphilis.

Summary

SUMMARY

- The most common age group affected with genital ulcer disease was 26-35 years -37%.
- Majority of the patients were unmarried – 61%
- 95% patients were uncircumcised.
- Heterosexual contact was most common – 64%
- Penovaginal intercourse was most common mode of sex – 70%.
- 84% patients had unprotected sexual intercourse.
- 65% patients presented with multiple ulcers.
- 98% patients had genital ulcers due to venereal etiology.
- Herpes genitalis was the most common cause of genital ulcers – 64.29%.
- Incidence of HIV was 15% and Hepatitis B was 4% and Hepatitis C was 1%.
- Coincidence of HIV, Hepatitis B and Hepatitis C in the study population with genital ulcer is 1%.

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Annexures

PROFORMA

"Coincidence of Hepatitis B,Hepatitis C and Retroviral disease with Genital ulcer among male patients attending STD OP"

Name : Age: Sex:

Occupation :

Education : Uneducated / Up to 5th / 12th / College

Address :

Marital Status : Single / Married / Separated / Divorced

History of sexual partner :

History of past or present genital ulcer in the partner :

History of drug abuse :

Referred by :

Presenting Complaints :

General Checkup / Screening

Genital Ulcer :

- Prior history of vesicles :
- Duration :
- Number :
- Recurrence :
- Pain :
- Associated with discharge :

History of Burning Micturition :

History of Urethral Discharge:

- Duration :
- Colour :

Swelling in Inguinal Region :

History of weight loss :

Frequent Diarrhea :

Persistent Fever :

Chronic cough for more than one month :

History of jaundice :

Treatment History :

Treatment taken for present complaints :

Exposure History :

- Recent exposure with dates :
- Pre marital contact :
- Extra marital contact :
- Last contact :

Usage of condom : Always/ Sometimes/ Never

Mode of Sex : Oral / Vaginal / Anal

Health Seeking Behaviour :

Awarness of STDs and Treatment :

Past History:

- Tuberclosis :
- Hypertension :
- Bronchial Asthma :
- Diabetes Mellitus :
- STDs :

Contact History:

Partner Name & Card No :

Occupation :

History of Alcohol / Smoking / I.V. drug abuse :

EXAMINATION:

GENERAL EXAMINATION:-

Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy / Pedal Edema

Pulse Rate :

Blood Pressure :

SYSTEMIC EXAMINATION:-

Cardio Vascular System :

Respiratory System :

Per Abdomen :

Central Nervous System :

GENITAL EXAMINATION:-

Inguinal Nodes :

External Urethral Meatus :

ULCER

Inspection:

Number :

Site :

Size :

Depth :

Floor : Necrotic Slough / Granulation Tissue / Clean

INFORMATION SHEET

- We are conducting a study on coincidence of HSV1,HSV2,HBV,anti HCV,syphilis and HIV in male patients with genital ulcer attending Rajiv Gandhi Government General Hospital, Chennai and your co-operation may be valuable to us.

- The purpose of this study is to estimate the seroprevalence of the sexually transmitted infections.

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

- All patients with genital ulcer attending op will be selected for this study.

Detailed clinical history (basic demographic details, presenting complaints, marital history, sexual history/ last contact, sexually transmitted infections in the past) followed by thorough clinical evaluation.

5 ml blood will be withdrawn aseptically from the participants. The serum will be separated and subjected to the tests for the detection of HIV ,Syphilis,HbsAg , anti HCV and HSV1 and HSV2.

Tzanck smear will be done in all patients. Scrapings from genital ulcer will be subjected to gram staining and wet mount examination. Dark field microscopy will be done from the serous exudates. Tissue smear will be done from the edge of the ulcer in relevant cases.

Signature of investigator

Signature of participant

Date:

PATIENT CONSENT FORM

Title of the study : **"Coincidence of Hepatitis B,Hepatitis C and Retroviral disease with Genital ulcer among male patients attending STD OP"**

Name of the participant :

Name of the principal investigator : Dr. V.Lakshmi Priya

Name of the Institution : Institute of Venereology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai – 3.

Documentation of the informed consent:

I ---------- have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me
2. I have had the consent document explained to me
3. I have been explained about the nature of the study
4. My rights and responsibilities have been explained to me by the investigator
5. I agree to co operate with the investigator and I will inform him/her immediately if I suffer unusual symptoms
6. I have not participated in any research study at any time
7. I am unaware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital
8. I hereby give permission to the investigator to release the information obtained from me as a result of participation in this study to the sponsors, regulatory authorities, Government agencies and institutional ethics committee. I understand that they are publicly presented.
9. My identity will be kept confidential if my data are publicly presented
10. I am aware that if I have any question during the study, I should contact at one of the addresses listed above. By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me, I will be given a copy of this consent document.

Participant initials:

For adult participants:

Name and signature/ thumb impression of the participant (or legal representative if participant incompetent)

_____	_____	----------
Name	Signature	Date

Name and signature of impartial witness (required for illiterate patients):

_____	_____	_____
Name	Signature	Date

Address and contact number of the impartial witnesss :

Name and signature of the investigator or his representative obtaining consent:

_____	_____	_____
Name	Signature	Date

சுய ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு : பால்வினை நோய் துறையின் வெளிநோயாளிகள் பிரிவிற்கு ஆண் உறுப்பில் புண்ணுடன் வரும் ஆண்களுக்கு Hepatitis B நோய்,Hepatitis C நோய் மற்றும் HIV நோய்களின் தற்செயல் இணைவு பற்றிய ஆய்வு.

பெயர் : வயது : தேதி : உள்நோயாளி எண் :

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக அறிந்து கொண்டேன். எனது சந்தேகங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன் பங்கு கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில்தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்து கொள்ளலாம் என சம்மதிக்கிறேன்.

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ வெளியிடப்பட்டாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்று கொண்டேன்.

இந்த ஆய்விற்காக ஆண் உறுப்பில் உள்ள புண்ணில் இருந்து நீர் எடுத்து Wet Mount, Gram Stain, Tzanck Smear, Dark Field, Tissue Smear போன்ற பரிசோதனைகளுக்கு உட்படுத்தப்பட்டு இரத்த மாதிரி பெறப்பட்டு (VCTC, VDRL, TPHA, HBsAg, Anti HCV, HSV1, HSV2) பரிசோதனைகளும் செய்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்றும் தெரிவிக்கிறேன் என்று புரிந்து கொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்றும் தெரிந்து கொண்டேன்.

பங்கேற்பாளர் / பாதுகாவலர் கையொப்பம் தேதி :

ஆய்வாளர் கையொப்பம் தேதி :

ஆய்வு தகவல் தாள்

ஆராய்ச்சியின் தலைப்பு : பால்வினை நோய் துறையின் வெளிநோயாளிகள் பிரிவிற்கு ஆண் உறுப்பில் புண்ணுடன் வரும் ஆண்களுக்கு Hepatitis B நோய் Hepatitis C நோய் மற்றும் HIV நோய்களின் தற்செயல் இணைவு பற்றிய ஆய்வு.

ஆய்வாளர் : மரு. வே. லட்சுமி பிரியா

பங்கேற்பாளர் : வயது :

ஆராய்ச்சி மையம் : பால்வினை நோய் துறை,
இராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

இந்த ஆய்வில் பங்கேற்பதற்காக தாங்கள் அழைக்கப்படுகிறீர்கள். இந்த ஆவணத்தில் உள்ள தகவல்கள் தாங்கள் இந்த ஆய்வில் பங்கேற்க முடிவு செய்துக் கொள்ள உதவும். இதில் ஏதேனும் சந்தேகம் இருந்தால் வெளிப்படையாக கேள்விகளைக் கேட்டு தெரிந்துக் கொள்ளலாம்.

நாங்கள் இராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் பால்வினை நோய் துறையின் வெளிநோயாளிகள் பிரிவிற்கு ஆண் உறுப்பில் புண்ணுடன் வரும் ஆண்களுக்கு Hepatitis B நோய் Hepatitis C நோய் மற்றும் HIV நோய்களின் தற்செயல் இணைவு பற்றிய ஆய்வை நடத்துகிறோம்.

அதற்கு உங்கள் பங்களிப்பு எங்களுக்கு பெரிதும் உதவக்கூடும்.

இந்த ஆய்வின் நோக்கம்:

இவ்வாராய்ச்சியில் தங்களிடையே அடிப்படை மற்றும் பாலியல் நோய் குறித்த விரிவான கேள்விகள் கேட்கப்படும். பின்னர் நீங்கள் மருத்துவ ரீதியான பிறப்புறுப்பின் பரிசோதனைக்கு உட்படுத்தப்படுவீர்கள்.

அனைவரிடமும் ஆண் உறுப்பில் உள்ள புண்ணில் இருந்து நீர் எடுத்து Wet Mount, Gram Stain, Tzanck Smear, Dark Field, Tissue Smear போன்ற பரிசோதனைகளுக்கு உட்படுத்தப்படுவீர்கள். அனைவரிடமும் இரத்த மாதிரி பெறப்பட்டு (VCTC, VDRL, TPHA, HBsAg, Anti HCV, HSV1, HSV2) பரிசோதனைகளும் செய்யப்படும்.

தங்களது மருத்துவ சிகிச்சை குறித்த தகவல்கள் இரகசியமாக பாதுகாக்கப்படும். ஆய்வின் போதோ அல்லது முடிவுகளை வெளியிடும் போதோ தங்களது பெயரையோ, அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை தெரிவித்துக் கொள்கிறோம்.

இந்த ஆய்வில் பங்கேற்பது உங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆய்விலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம். இந்த ஆய்வில் பங்கேற்காவிட்டாலும் நீங்கள் வழக்கமான சிகிச்சையை தொடர்ந்து பெறலாம்.

இந்த ஆய்வின் முடிவு தங்களுக்கு ஆய்வின் இறுதியிலோ அல்லது ஆய்வின் போதிலோ தெரியப்படுத்தப்படும்.

ஆய்வாளர் கையொப்பம்

பங்கேற்பாளர் / பாதுகாவலர்
கையொப்பம்

தேதி :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301A
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.V. Lakshmi Priya
Post Graduate in MD DVL
Madras Medical College
Chennai 600 003

Dear Dr.V.Lakshmi Priya,

The Institutional Ethics Committee has considered your request and approved your study titled **"COINCIDENCE OF HEPATITIS B,HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD OP" NO. 21112016.**

The following members of Ethics Committee were present in the meeting hold on **01.11.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.A.Rajendran,MS, Prof. of Surgery,MMC,Ch-3	: Member
6.Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
7.Prof.Baby Vasumathi,MD.,Director, Inst. of O & G	: Member
8.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
9.Prof.R.Padmavathy, MD, Director,Inst.of Pathology,MMC,Ch-3	: Member
10.Prof.S.Mayilvahanan,MD,Director, Inst. of Int.Med,MMC, Ch-3	: Member
11.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE.
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

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CERTIFICATE –II

This is to certify that this dissertation work titled **"COINCIDENCE OF HEPATITIS B, HEPATITIS C AND RETROVIRAL DISEASE WITH GENITAL ULCER AMONG MALE PATIENTS ATTENDING STD O.P."** of the candidate **Dr.V.LAKSHMI PRIYA,** with registration Number **201530004** for the award of **M.D DERMATOLOGY, VENEREOLOGY & LEPROSY** in the branch of **XX**. I personally verified the urkund .com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **1 percentage** of plagiarism in the dissertation.

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MASTER CHART

S. No.	Age	literacy	Marital status	Sexual orientation	Last contact	Mode of sex	Number of ulcers	Pain	Nodes	Tzanck smear	Grams stain	KOH	Normal Saline	VCTC	HBS Ag	anti HCV ab	VDRL	TPHA	protection	PVD	circumcision
1	51	5th	married	heterosexual	6 months	penovaginal	single	absent	present	negative	pus cells	negative	pus cells	positive	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
2	43	10 th	unmarried	heterosexual	10 days	penovaginal	multiple	present	present	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	circumcised
3	36	diploma	married	heterosexual	4 months	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
4	27	10 th	unmarried	homosexual	6 months	anoinsertive	multiple	present	absent	positive	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
5	30	degree	unmarried	homosexual	3 months	oroinsertive	single	absent	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
6	29	BSc	unmarried	heterosexual	2 months	penovaginal	multiple	absent	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
7	28	8 th	unmarried	bisexual	1 month	penovaginal	multiple	absent	absent	positive	pus cells	negative	pus cells	negative	negative	negative	1:64 reactive	positive	unprotected	absent	circumcised
8	18	BBA	unmarried	homosexual	2 months	anoinsertive	multiple	absent	absent	negative	negative	negative	negative	negative	positive	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
9	37	IT	married	bisexual	1 month	penovaginal	single	present	present	negative	negative	negative	negative	positive	negative	negative	1:128 reactive	positive	unprotected	absent	uncircumcised
10	40	8 th	unmarried	heterosexual	6 days	penovaginal	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
11	30	12 th	unmarried	bisexual	6 months	penovaginal	multiple	present	present	negative	pus cells	negative	negative	positive	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
12	38	degree	married	bisexual	1 month	ororecptive	multiple	absent	absent	positive	negative	negative	negative	positive	negative	negative	non reactive	negative	unprotected	present	uncircumcised
13	49	diploma	married	heterosexual	1 month	penovaginal	multiple	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
14	30	illiterate	unmarried	heterosexual	6 months	penovaginal	single	absent	absent	negative	epithelial cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
15	28	illiterate	unmarried	bisexual	20 days	oroinsertive	multiple	present	present	negative	negative	negative	negative	negative	negative	negative	1:16 reactive	positive	unprotected	present	uncircumcised
16	34	ug	married	heterosexual	3 months	penovaginal	multiple	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
17	22	ug	unmarried	heterosexual	1 week	penovaginal	single	absent	absent	negative	negative	negative	negative	negative	negative	negative	1:2 reactive	positive	unprotected	present	uncircumcised
18	33	degree	married	bisexual	4 weeks	oroinsertive	single	present	absent	negative	negative	negative	negative	negative	negative	negative	1:8 reactive	positive	unprotected	absent	uncircumcised
19	20	ug	unmarried	heterosexual	45 days	penovaginal	single	absent	absent	negative	pus cells	negative	negative	negative	positive	negative	1:64 reactive	positive	unprotected	present	uncircumcised
20	29	degree	unmarried	heterosexual	2 years	penovaginal	multiple	present	present	negative	pus cells	negative	negative	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
21	36	degree	married	heterosexual	5 months	penovaginal	multiple	absent	absent	positive	negative	negative	negative	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
22	23	10 th	unmarried	heterosexual	1 month	penovaginal	multiple	absent	present	negative	negative	negative	negative	negative	negative	negative	1:32 reactive	positive	unprotected	absent	uncircumcised
23	47	illiterate	married	heterosexual	1 week	penovaginal	single	present	present	negative	pus cells	negative	pus cells	positive	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
24	48	illiterate	married	bisexual	1 month	anoinsertive	multiple	absent	present	negative	negative	negative	negative	negative	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
25	26	12 th	unmarried	heterosexual	1 month	penovaginal	multiple	present	present	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	protected	absent	uncircumcised
26	31	degree	married	heterosexual	1 week	penovaginal	single	present	present	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
27	51	illiterate	married	heterosexual	8 yrs	penovaginal	single	present	absent	negative	pus cells	negative	pus cells	positive	negative	negative	non reactive	negative	unprotected	present	uncircumcised
28	26	diploma	married	heterosexual	2 weeks	penovaginal	single	absent	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
29	36	12 th	married	heterosexual	10 days	penovaginal	multiple	present	present	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
30	24	10 th	unmarried	heterosexual	6 months	penovaginal	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
31	43	8 th	married	heterosexual	1 week	penovaginal	single	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
32	52	8 th	unmarried	heterosexual	2 weeks	penovaginal	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
33	35	9 th	married	heterosexual	1 month	penovaginal	multiple	present	absent	negative	pus cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
34	22	degree	unmarried	homosexual	6 months	anoreceptive	single	absent	present	negative	pus cells	negative	pus cells	negative	positive	negative	1:4 reactive	positive	unprotected	present	uncircumcised
35	42	6 th	married	heterosexual	2 months	penovaginal	multiple	absent	present	positive	pus cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
36	29	degree	unmarried	homosexual	3 weeks	anoinsertive	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	protected	present	uncircumcised

S. No.	Age	literacy	Marital status	Sexual orientation	Last contact	Mode of sex	Number of ulcers	Pain	Nodes	Tzanck smear	Grams stain	KOH	Normal Saline	VCTC	HBS Ag	anti HCV ab	VDRL	TPHA	protection	PVD	circumcision
37	56	5 th	married	heterosexual	3 months	penovaginal	multiple	absent	present	negative	pus cells	negative	pus cells	positive	negative	negative	non reactive	negative	unprotected	present	uncircumcised
38	25	illiterate	unmarried	homosexual	1 week	anoinsertive	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
39	40	illiterate	unmarried	homosexual	1 month	anoinsertive	multiple	present	absent	positive	pus cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
40	23	degree	unmarried	heterosexual	5 days	penovaginal	single	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
41	33	illiterate	unmarried	bisexual	20 days	anoreceptive	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:2 reactive	positive	unprotected	present	uncircumcised
42	28	degree	unmarried	homosexual	1 month	anoreceptive	single	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
43	25	diploma	unmarried	homosexual	45 days	anoinsertive	single	absent	absent	negative	negative	negative	negative	negative	negative	negative	1:128 reactive	positive	unprotected	present	uncircumcised
44	32	11 th	married	heterosexual	2 weeks	penovaginal	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	protected	absent	uncircumcised
45	39	degree	married	heterosexual	5 months	penovaginal	single	absent	absent	negative	negative	negative	negative	positive	negative	negative	non reactive	negative	unprotected	present	uncircumcised
46	54	degree	married	heterosexual	45 days	penovaginal	multiple	absent	present	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
47	48	diploma	married	heterosexual	1 year	penovaginal	multiple	present	absent	positive	pus cells	negative	pus cells	positive	negative	negative	1:8 reactive	positive	unprotected	absent	uncircumcised
48	45	5 th	married	heterosexual	6 months	penovaginal	single	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
49	62	illiterate	married	heterosexual	4 months	penovaginal	multiple	absent	absent	negative	pus cells	negative	pus cells	negative	negative	negative	1:16 reactive	positive	unprotected	absent	uncircumcised
50	20	5 th	unmarried	homosexual	1 month	anoinsertive	single	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
51	28	6 th	unmarried	homosexual	3 months	anoreceptive	multiple	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
52	25	degree	unmarried	heterosexual	2 months	penovaginal	single	present	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
53	31	degree	married	bisexual	3 months	penovaginal	multiple	absent	absent	positive	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
54	35	degree	married	heterosexual	1 month	penovaginal	multiple	absent	absent	negative	negative	negative	negative	negative	negative	negative	1:32 reactive	positive	unprotected	present	uncircumcised
55	29	diploma	unmarried	homosexual	15 days	anoreceptive	single	absent	absent	negative	pus cells	negative	negative	negative	negative	negative	1:32 reactive	positive	unprotected	present	uncircumcised
56	23	9 th	unmarried	bisexual	10 days	anoinsertive	multiple	present	absent	negative	pus cells	negative	pus cells	positive	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
57	29	10 th	unmarried	heterosexual	4 days	anoinsertive	multiple	present	present	negative	pus cells	negative	negative	positive	positive	positive	1:16 reactive	positive	unprotected	present	uncircumcised
58	26	5 th	married	bisexual	1 week	penovaginal	multiple	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
59	18	illiterate	unmarried	heterosexual	2 weeks	penovaginal	multiple	absent	absent	positive	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
60	64	illiterate	married	heterosexual	4 months	penovaginal	multiple	present	absent	positive	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
61	28	degree	unmarried	bisexual	2 weeks	penovaginal	multiple	present	present	pus cells	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
62	26	5 th	unmarried	heterosexual	2 months	penovaginal	single	absent	present	negative	negative	negative	negative	negative	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
63	26	degree	unmarried	heterosexual	3 months	penovaginal	single	absent	present	negative	negative	negative	negative	negative	negative	negative	1:8 reactive	positive	unprotected	present	uncircumcised
64	60	5 th	married	heterosexual	5 yrs	penovaginal	multiple	present	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:16 reactive	positive	unprotected	present	uncircumcised
65	30	6 th	unmarried	heterosexual	2 months	penovaginal	single	absent	absent	negative	pus cells	negative	pus cells	positive	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
66	28	degree	unmarried	heterosexual	3 months	penovaginal	multiple	present	absent	pus cells	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
67	36	diploma	unmarried	heterosexual	4 months	penovaginal	multiple	absent	absent	negative	negative	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
68	25	degree	unmarried	heterosexual	3 months	penovaginal	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:128 reactive	positive	unprotected	present	uncircumcised
69	33	degree	unmarried	homosexual	15 days	anorecptive	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
70	40	degree	married	heterosexual	15 days	penovaginal	multiple	present	absent	negative	pus cells	negative	negative	negative	negative	Negative	non reactive	negative	unprotected	present	circumcised
71	38	degree	unmarried	heterosexual	I month	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	circumcised
72	65	5 th	married	heterosexual	3weeks	penovaginal	multiple	present	absent	negative	pus cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
73	26	degree	unmarried	bisexual	1 week	anoinsertive	multiple	present	present	positive	pus cells	negative	pus cells	positive	negative	negative	non reactive	negative	unprotected	present	uncircumcised

S. No.	Age	literacy	Marital status	Sexual orientation	Last contact	Mode of sex	Number of ulcers	Pain	Nodes	Tzanck smear	Grams stain	KOH	Normal Saline	VCTC	HBS Ag	anti HCV ab	VDRL	TPHA	protection	PVD	circumcision
74	29	10 th	unmarried	heterosexual	1 month	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
75	40	5 th	married	heterosexual	2 months	penovaginal	multiple	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
76	44	illiterate	married	heterosexual	1 week	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
77	22	degree	unmarried	homosexual	10 days	anoinsertive	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
78	44	illiterate	married	heterosexual	2 weeks	penovaginal	multiple	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
79	31	illiterate	unmarried	heterosexual	1 month	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
80	19	ug	unmarried	homosexual	1 week	anoinsertive	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	absent	uncircumcised
81	24	diploma	unmarried	homosexual	1 month	anoinsertive	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:128 reactive	positive	unprotected	absent	uncircumcised
82	48	10 th	married	bisexual	15 days	penovaginal	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
83	23	degree	unmarried	heterosexual	15 days	penovaginal	single	absent	present	negative	pus cells	negative	negative	negative	negative	negative	1:16 reactive	positive	unprotected	present	uncircumcised
84	26	degree	unmarried	heterosexual	1 week	penovaginal	single	absent	absent	negative	negative	negative	pus cells	negative	negative	negative	1:8 reactive	positive	unprotected	present	uncircumcised
85	21	5 th	unmarried	heterosexual	1 month	penovaginal	single	absent	present	negative	pus cells	negative	negative	negative	negative	negative	1:64 reactive	positive	unprotected	present	uncircumcised
86	26	degree	unmarried	homosexual	2 weeks	anoreceptive	single	absent	present	negative	pus cells	negative	pus cells	positive	negative	negative	1:64 reactive	positive	unprotected	absent	uncircumcised
87	23	degree	unmarried	homosexual	1 week	anoinsertive	single	absent	present	negative	pus cells	negative	pus cells	positive	negative	negative	1:64 reactive	positive	unprotected	present	uncircumcised
88	44	10 th	married	heterosexual	2 weeks	penovaginal	multiple	present	present	positive	pus cells	negative	negative	negative	negative	negative	1:16 reactive	positive	unprotected	present	uncircumcised
89	39	7 th	married	heterosexual	2 weeks	penovaginal	multiple	absent	absent	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
90	26	degree	unmarried	heterosexual	1 month	penovaginal	multiple	present	absent	negative	pus cells	negative	negative	negative	negative	negative	non reactive	negative	unprotected	present	circumcised
91	32	degree	unmarried	heterosexual	3 months	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
92	23	diploma	unmarried	heterosexual	4 months	penovaginal	multiple	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	protected	present	uncircumcised
93	23	degree	unmarried	heterosexual	1 month	penovaginal	multiple	absent	absent	negative	negative	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
94	23	degree	unmarried	homosexual	2 months	anoinsertive	multiple	present	absent	positive	negative	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
95	50	10 th	married	heterosexual	3months	penovaginal	multiple	present	absent	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	absent	uncircumcised
96	23	degree	unmarried	heterosexual	3 months	penovaginal	multiple	present	absent	negative	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
97	25	degree	unmarried	homosexual	2 months	anorecptive	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:64 reactive	positive	unprotected	present	uncircumcised
98	20	12 th	unmarried	homosexual	10 days	anoinsertive	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
99	46	10 th	married	heterosexual	1 month	penovaginal	multiple	present	present	positive	pus cells	negative	pus cells	negative	negative	negative	non reactive	negative	unprotected	present	uncircumcised
100	21	illiterate	unmarried	homosexual	1 week	anoinsertive	single	absent	present	negative	pus cells	negative	pus cells	negative	negative	negative	1:64 reactive	positive	unprotected	present	uncircumcised

KEY FOR MASTER CHART

1.	KOH	-	Potassium Hydroxide
2.	VCTC	-	Voluntary Counselling and Testing Center
3.	HBSAg	-	Hepatitis B Surface Antigen
4.	VDRL	-	Venereal Disease Research Laboratory
5.	TPHA	-	Treponema Pallidum Hemagglutination
6.	PVD	-	Previous Venereal Disease

KEY FOR REVIEW OF LITERATURE

1. AIDS - Acquired Immuno Deficiency Syndrome
2. ART - Anti Retroviral Therapy
3. AST - Aspartate Transaminase
4. ALT - Alanine Transaminase
5. CD - Cluster of Differentiation
6. CNS - Central Nervous System
7. CCR - Chemokine CoReceptor
8. DNA - Deoxyribo Nucleic Acid
9. GIT - Gastro Intestinal Tract
10. GUD - Genital Ulcer Disease
11. HCV - Hepatitis C Virus
12. HBSAg - Hepatitis B Surface Antigen
13. HIV - Human Immunodeficiency Virus
14. HAART- Highly Active Anti Retroviral Therapy
15. IRIS - Immune Reativation Inflammatory Syndrome
16. LGV - Lympho Granuloma Venereum
17. MSM - Men having Sex with Men
18. NHL - Non Hodgkins Lymphoma
19. NSAID - Non Steroidal Anti Inflammatory Drug
20. PCR - Polymerase Chain Reaction
21. RNA - Ribo Nucleic Acid
22. TB - Tuberculosis